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THE
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VOL. LXIII JUNE, 1953 No. 6

HEARING LOSSES FOLLOWING PARTIAL SECTION OF THE COCHLEAR NERVE.*†‡

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(by invitation)

INTRODUCTION.

Some of the effects on the morphology and function of the cochlea resulting from degeneration of the cochlear nerve are known. Wittmaack,[1] in 1911, reported that division of the cochlear nerve results in irreversible degenerationof the spiral ganglion, but that the organ of Corti remains normal. Kaida,[2] as well as Hallpike and Rawdon Smith,[3] confirmed this finding and demonstrated in addition that to maintain the integrity of the inner ear structures it is necessary to preserve the cochlear artery.

Dandy[4] partially sectioned the VIIIth cranial nerve in human patients for the relief of Ménière's disease. He often found no change in auditory thresholds even though large portions of the cochlear nerve were sacrificed. Hearing losses that did occur were for high frequencies. Neff[5] studied the effect on auditory threshold of partial section of the cochlear nerve in conditioned cats. He also found that severe nerve lesions were compatible with normal auditory thresholds.

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Several of the animals, however, sustained high frequency hearing losses, which was in accordance with Dandy's experience in human patients. He noted that any threshold losses produced were always for high frequencies even though different portions of the nerve were selectively sectioned. This finding led Neff to raise the question as to whether nerve fibres which respond selectively to low frequency stimuli actually exist. The present investigation is an attempt to provide further information relative to this question by selective section of cochlear nerve fibres in animals conditioned for auditory testing.

PROCEDURE.

For each of the six cats the function of one ear was destroyed by macerating the labyrinth with a probe introduced through the round window. The animals were then trained to respond to pure tone auditory stimuli by moving forward in a rotating cage to avoid shock. Audiograms were determined, using this conditioned avoidance response as an indicator of hearing. The method is described in detail elsewhere.[6]

In all cats, except Cat 4, operations were then done to obliterate either the cochlear aqueduct or endolymphatic sac. In Cats 1, 3 and 5 both procedures were performed with an interval of several months between operations. In none of these animals did the auditory thresholds change from the preoperative levels as a result of these operations. Thus it seemed fairly certain that the sensory and neural elements were still normal in these ears. A detailed report on that part of the experimentation relating to obliteration of the cochlear aqueduct and endolymphatic sac is given elsewhere.[7]

Because of the investment of time and energy in conditioning of these animals, the decision was made to use the animals for an experiment on partial section of the cochlear nerve. Thus, after recovery periods varying from two to seven months the animals were subjected to an operation for partial section of the cochlear nerve.

A curved incision was made through the scalp over the parietal and occipital bones near the midline. The temporalis

muscle was detached from the parietal and temporal bones and reflected laterally and forward. The dorsal extensor mus-

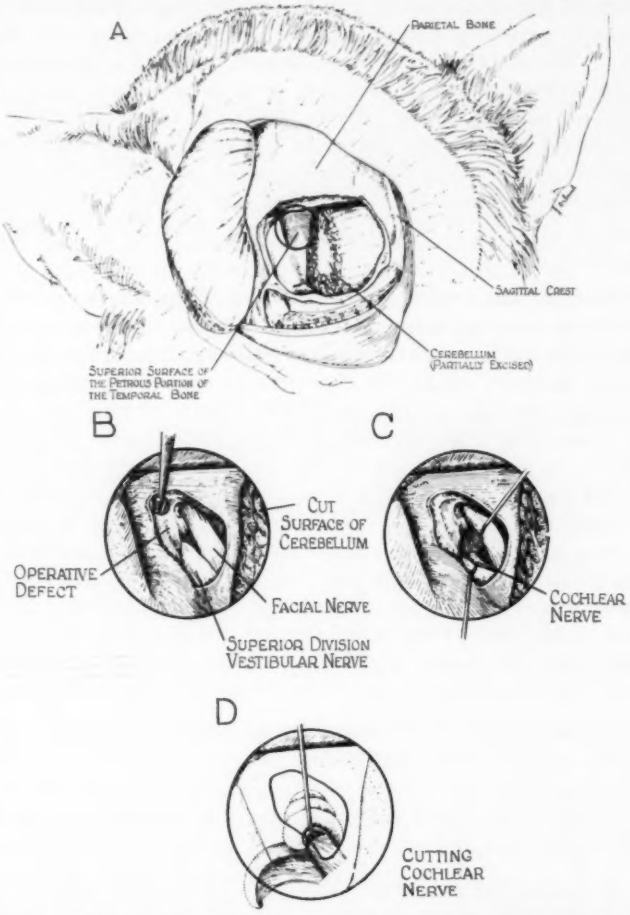

Fig. 1. Operative technique. (A) Exposure of petrous pyramid after removal of part of cerebellum. (B) Bone removed to expose the facial nerve and superior division of the vestibular nerve. (C) Retraction of facial nerve and superior division of the vestibular nerve to expose cochlear nerve as it enters the modiolus. (D) Diagrammatic drawing showing selective section of cochlear nerve fibres supplying the upper half of the cochlea. In reality this was only partially accomplished.

cles of the head were detached from the lamboidal ridge and reflected caudally. Portions of the parietal, temporal and occipital bones, including the lamboidal ridge, were removed so as to expose widely the posterior cranial fossa (see Fig. 1A). The dura was removed. About one-half of the underlying cerebellar hemisphere was removed so as to expose the superior surface of the petrous portion of the temporal bone. In several animals the tentorium was removed and the occipital lobe of the cerebellum was retracted forward gently so as to provide adequate exposure of the petrous bone. Absorbable gelatin sponge, U. S. P. (gelfoam®), was used to control bleeding.

Using a bone engine and small burrs, bone was removed in an ovoid area anteromedial to the arcuate eminence (superior semicircular canal) and immediately superior to the internal auditory canal. In this manner the facial nerve and superior division of the vestibular nerve were exposed to view (see Fig. 1B). By retracting the facial nerve anteriorly and the superior division of the vestibular nerve posteriorly it was possible to expose the cochlear nerve as it entered the modiolus (see Fig. 1C). The nerve was sectioned by gently moving a needle through the nerve trunk at this point (see Fig. 1D). It was thought that this technique would sever the fibres supplying the apical and middle turns and the upper part of the basal turn while leaving the fibres to the lower basal turn and "hook" region intact. The histological findings show that, in reality, this was only partially accomplished.

The temporalis muscle was sutured into place over the skull defect, and the scalp was closed.

All animals were given penicillin intramuscularly for five days postoperatively in doses adequate to maintain an effective blood level.

All operations were performed with the animals under deep anethesia with intraperitoneally injected pentobarbital (nembutal®) sodium.

Beginning one week after operation, tests of hearing were made at intervals of three to seven days for each animal. The

auditory thresholds showed no significant change in the interval between the first test made one week postoperatively and the last tests performed about four weeks postoperatively. The final audiograms were based on several tests taken on consecutive days.

About one month after the nerve section each animal was anesthetized and subjected to a cortical test of auditory function. This test method has been described in detail by Hind.[8,9] In brief, the method consisted of treating the auditory cortex with strychnine sulfate solution so that spikelike waves of electrical potential could be recorded in response to the relatively abrupt onset of pure tones. A determination was made of the sound pressure level required for threshold at all frequencies of interest. These threshold intensities were compared with the average values obtained for a group of normal animals.

The purpose of performing the cortical test in these animals was to determine the reliability of the method as a test of peripheral auditory function. In general, the cortical test results agree with the behavioral tests and the data are presented because of the support they give to the evaluation of cochlear function in these animals. Thus, two curves appear on the graph for each ear: the behavioral in terms of the difference between pre- and postoperative thresholds, and the cortical in terms of the difference between the average thresholds for normal cats and the thresholds for the particular animal.

In a previous publication experimental evidence was presented which demonstrated approximate locations on the organ of Corti of the regions of maximum excitation for various frequencies of the auditory spectrum of the cat.[6] Thus, for example, the region of maximum excitation for 32,000 c.p.s. was 9 per cent of the distance along the organ of Corti as measured from the basal end, 16,000 c.p.s. was at 21.3 per cent, 8,000 c.p.s. at 33.6 per cent, etc. These values form the basis for the distribution of frequencies on the abscissa of the audiogram, which is termed the anatomical frequency scale.

Microphonic and neural responses to sustained tones and clicks also were recorded from the ears of Cats 1, 2, 5 and 6. The electrode consisted of platinum foil which was placed on the margin of the round window membrane. It was attached to platinum wire led out through a small hole in the auditory bulla. The wire was placed against the margin of the bulla opening, and the opening was sealed with dental cement.

Immediately following these tests the animals were sacrificed by arterial perfusion with Heidenhain Susa solution. The inner ears were serially sectioned at a thickness of 20 and every fifth section stained with hematoxylin and eosin and mounted for study. Additional sections were prepared as needed for detailed study. Several nerve fibre stains were made of each ear.

The organ of Corti and spiral ganglion of each ear were graphically reconstructed. Distance along the organ of Corti was indicated in millimeters, beginning at the basal end. Points along the spiral ganglion having a radial relationship to the millimeter points on the organ of Corti were given the same numerical designations (see Fig. 2).

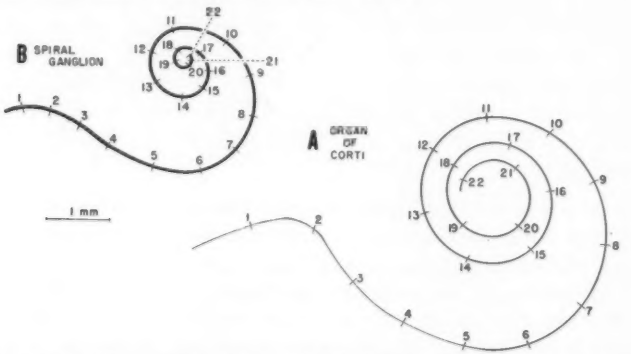

Fig. 2. Graphic reconstructions. (A) Spiral representing the line of contact of the heads of the pillar cells. Distance is indicated in millimeters. (B) From the same ear a spiral representing a line passing through the center of the spiral ganglion. The numbers indicate points having a radial relationship to the millimeter points along the organ of Corti.

It is known that cutting the axone of the spiral ganglion cell causes total and permanent degeneration of that cell;

therefore, the change in the spiral ganglia of the ears under study is presented in terms of the percentage of ganglion cells remaining after degeneration of injured ganglion cells has occurred.

Ganglion cells were made from 16 to 20 areas distributed throughout the course of the spiral ganglion according to the following method: The region of the spiral ganglion to be studied was placed under an eyepiece counting grid (see Fig. 3A). A copy of the grid was made on ruled paper and the outstanding landmarks appearing on the microscopic section, such as bone margins and blood vessels, were placed in the drawing so that a particular grid placement could be reproduced if desired. The location of neurons lying at the periphery of the ganglion was noted on the counting grid and these cells were sketched into their proper locations in the drawing. A line connecting these cells formed the boundary of the area from which the ganglion cell counts were made. All ganglion cells in which a nucleolus was clearly visible were counted. From the size of the counting grid and the thickness of the microscopic section it was a simple matter to calculate the number of ganglion cells per unit volume of space in Rosenthal's canal. An absolute count should include only one-half of the nucleoli which have been split by the microtome knife. It is not possible to determine with certainty from the microscopic appearance whether a nucleolus has been split by sectioning. The per cent of split nucleoli should be constant for all ears, however. Thus, the values obtained from the ganglion cell counts in the pathological ears have been expressed in terms of per cent of normal, which obviates the necessity for introducing a correction factor for split nucleoli.

The ganglion cell counts from three presumably normal cat ears appear in Fig. 3C. There are about 145 cells per 0.001 c.mm. of Rosenthal's canal for the part of the ganglion supplying the organ of Corti, located at 5 per cent of its length as measured from the basal end. The count rises progressively to 200 at 20 per cent of the distance and remains at about that figure in the remainder of the ganglion. In the upper 80 per cent of the cochlea most of the counts fell in the range

between 170 and 230 cells per 0.001 c.mm. For one ear the counts were about 30 cells higher throughout than for the other two ears. In one ear the count was 248 at 57 per cent of the distance and 170 at 64 per cent, a variation in the count

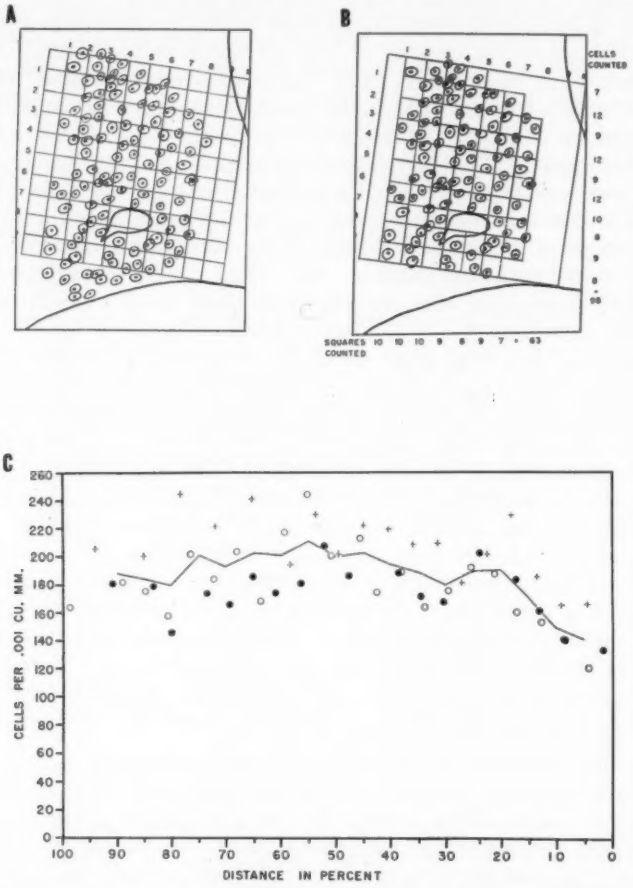

Fig. 3. (A) Diagram demonstrating placement of counting grid on cut section of spiral ganglion. (B) Diagram showing that 98 cells were counted in 63 squares. The two squares containing the vessel were excluded from the count. (C) Ganglion cell counts from the normal cat ears. The line represents the average values.

of 78 cells in a distance range of 7 per cent; furthermore, in the 80 per cent region of the ear of Cat N-1 the count was 250, whereas in approximately the same region in Cat N-2 the count was 160. A continuous line has been drawn in the graph of Fig. 3C to indicate average normal as used in this study. Obviously the spread of normal values is so large that small deviations from the average are of no significance.

RESULTS.

Cat 1: The auditory thresholds were normal following operations to destroy the endolymphatic sac and obliterate the cochlear aqueduct in the experimental ear. Three months later an operation was performed to partially section the left cochlear nerve according to the method described. Following this operation, the behavioral threshold for 8,000 c.p.s and 16,000 c.p.s. remained normal. There was a loss of 39 db for 4,000 c.p.s., 25 db. for 2,000 c.p.s., 55 db for 1,000 c.p.s., 58 db for 500 c.p.s., and 62 db for 250 c.p.s. The frequencies 62.5 and 125 c.p.s were not heard at maximum intensities of 50 db and 55 db, respectively, above preoperative thresholds (see Fig. 4).

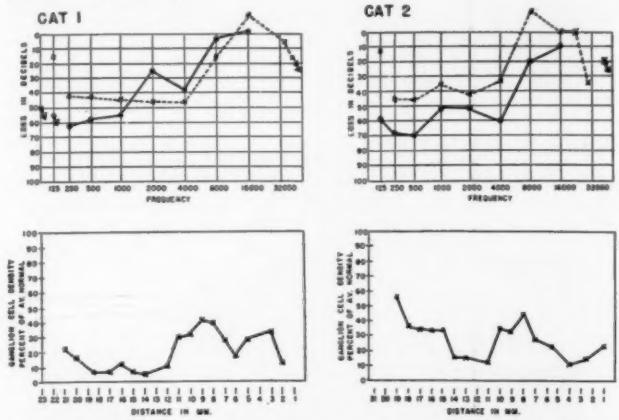

Fig. 4. In the upper chart for each animal appear the behavioral test (continuous line) and cortical test (broken line) of auditory function. The behavioral test is plotted as gain or loss in sensitivity from the animal's preoperative threshold. The cortical test is plotted as the gain or loss in sensitivity as compared to the average normal threshold for healthy cats. Frequencies on the abscissa are located in accordance with the anatomical frequency scale. In the lower chart the per cent of spiral ganglion cells remaining is plotted as a function of distance as measured along the organ of Corti (see text for explanation). Cat 1 was sacrificed one month after partial section of the cochlear nerve. Cat 2 was sacrificed three and one-half weeks after such an operation.

One month after the nerve section a cortical test of auditory function was made. Three thresholds by the cortical test were elevated 40 to 50 db for frequencies from 250 c.p.s. to 4,000 c.p.s. The threshold was elevated

15 db for 8,000 c.p.s., was essentially normal for 16,000 c.p.s. and 35,000 c.p.s., and again elevated 16 db for 40,000 c.p.s. There was no response to 45,000 c.p.s. delivered at an intensity of 20 db above average normal threshold. The frequency 125 c.p.s. showed a loss of only 15 db.

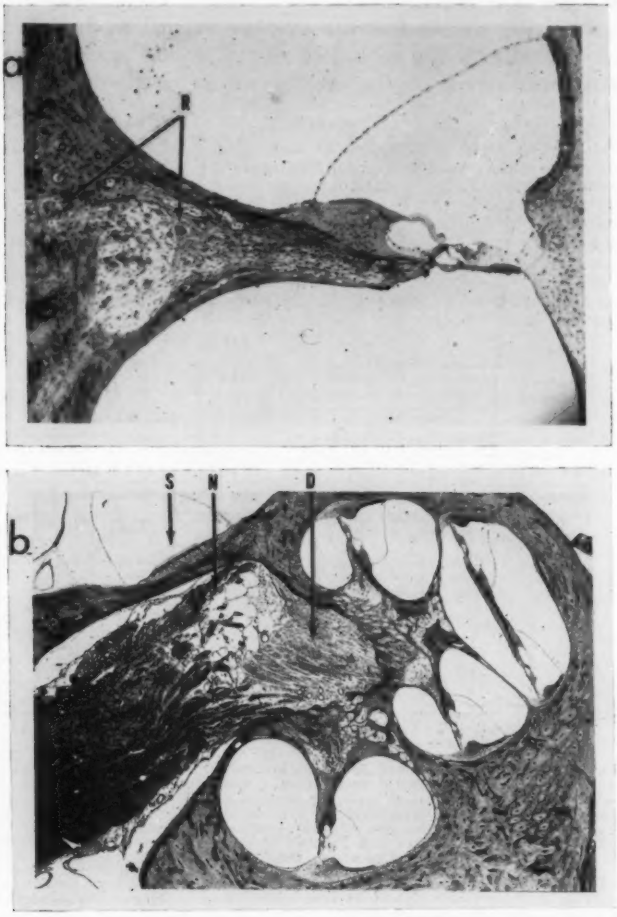

Fig. 5. (A) Photomicrograph from the 6 mm. area of the cochlea of Cat 1 showing a normal organ of Corti in an area in which only 23 per cent of the spiral ganglion cells remain. (R) is Rasmussen's bundle. (B) Cochlea of Cat 1. (S) is the saccule. (N) is the saccular nerve. (D) indicates degenerated cochlear nerve fibres.

Histopathology: Histological examination showed the endolymphatic sac to be destroyed and the lumen of the cochlear aqueduct to be obliterated by new bone and connective tissue.

Reissner's membrane bulged moderately into the scala vestibuli. The organ of Corti was normal throughout (see Fig. 5A). The most striking change in the cochlea was the loss of a great many spiral ganglion cells and their nerve fibres (see Figs. 5A and B). Ganglion cell counts were made at 17 points along the course of the ganglion and in Fig. 4 were plotted in terms of percentage of average normal population density. In the basal one-half of the cochlea about 20 to 40 per cent of ganglion cells remained, whereas in the apical one-half, only 5 to 20 per cent of these cells were still present.

The facial nerve and the nerve to the superior horizontal canal cristae were totally degenerated. Rasmüssen's (Olivo-cochlear) bundle was intact.

Cat 2: The auditory thresholds were unchanged over a four-month period following destruction of the endolymphatic sac. An operation was then performed to accomplish partial section of the cochlear nerve. Following this operation there was a hearing loss by the behavioral test of 50 to 70 db for frequencies from 125 c.p.s. to 4,000 c.p.s. and losses of 20 db for 8,000 c.p.s. and 10 db for 16,000 c.p.s.

Three and one-half weeks following section of the nerve, a cortical test of auditory function was made, following which the animal was sacrificed.

The cortical test showed threshold elevations of 35 to 45 db for frequencies from 250 c.p.s. to 4,000 c.p.s. The thresholds were normal for 8,000 c.p.s., 16,000 c.p.s. and 25,000 c.p.s. The threshold for the frequency 30,000 c.p.s. was elevated 34 db and there was no response to 40,000 c.p.s. at an intensity of 20 db above average normal threshold. The frequency 125 c.p.s. showed a loss of only 12 db.

Histopathology: Histologic study showed replacement of the endolymphatic sac by scar tissue.

The organ of Corti, Riessner's membrane and other structures of the cochlear duct appeared normal.

There was a marked loss in spiral ganglion cells throughout the cochlea. The population density in terms of percentage of average normal was 23 per cent of normal at 1 mm.; diminished to 12 per cent at 4 mm., and then rose steadily to 45 per cent at 8 mm. (see Fig. 4). The count then fell to reach a low of 13 per cent at 11 mm., and between 11 and 14 mm. ranged between 13 per cent and 17 per cent of normal. Between 15 and 18 mm. the values were between 32 and 37 per cent of normal and the last determination at 19 mm. was 57 per cent of normal.

The vestibuli and facial nerves were intact. The membranous vestibular labyrinth appeared normal.

Rasmüssen's bundle was intact.

Cat 3: No changes in auditory thresholds were noted over a period of two months following obliteration of the cochlear aqueduct and endolymphatic sac. An operation for partial section of the left cochlear nerve was then performed. Following the latter procedure, behavioral hearing tests showed a threshold elevation of 16 db for the 4,000 c.p.s. frequency, whereas the thresholds for the other frequencies remained essentially normal (see Fig. 6). Three weeks after the nerve section a cortical test of auditory function was performed, following which the animal was sacrificed.

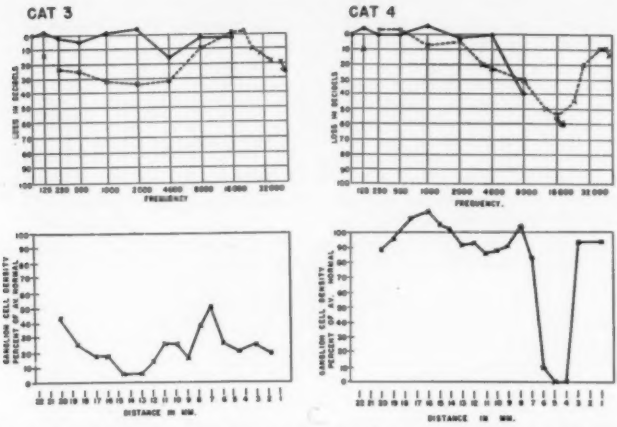

Fig. 6. Graphs of cochlear function and spiral ganglion degeneration for Cats 3 and 4. The continuous line is the behavioral audiogram and the broken line is the cortical test of auditory function. Cat 3 was killed three weeks after partial section of the cochlear nerve trunk. Cat 4 was killed one month following an operation in which a needle was introduced through the round window membrane into the osseous spiral lamina injuring spiral ganglion of the basal turn.

The cortical test revealed a rather flat 25 to 35 threshold elevation for frequencies 250 c.p.s. to 4,000 c.p.s. The thresholds for the frequencies 8,000 c.p.s., 16,000 c.p.s. and 20,000 c.p.s. were essentially normal. Above 20,000 c.p.s. there was a progressive elevation of threshold to 19 db for 35,000 c.p.s. There was no cortical response to the frequency 40,000 c.p.s. at an intensity of 20 db above average normal threshold. The frequency 125 c.p.s. showed a loss of only 14 db.

Histopathology: Histological examination showed that the operation to obliterate the cochlear aqueduct was unsuccessful as the structure was normal in appearance. The endolymphatic sac was missing, and the space was filled with scar tissue. The bony canal of the crus commune had been injured and was partially filled with new bone; however, the membranous canal was intact in this region.

The structures within the cochlear duct, including the hair cells, appeared normal. There was a small amount of blood in the scala tympani and scala vestibuli in the most inferior aspects of all three turns.

There was a diffuse degenerative change in the spiral ganglion. Ganglion cell counts revealed only 20 to 25 per cent of the cells to be present in the region between 2 and 6 mm. At 7 mm., 50 per cent of the cells remained, but from 7 mm., the count decreased progressively to a low of 5 per cent at 13 and 14.5 mm. Apicalward from 14.5 mm. the population density again increased progressively to a high of 43 per cent of normal at 20 mm.

The nerve bundles to the superior and horizontal canal ampuli were almost totally degenerated. The branch to the saccular macula was

normal. The facial nerve and inferior division of the vestibular nerve were normal. The membranous vestibular labyrinth appeared normal.

Rasmüssen's bundle was intact.

Cat 4: A lesion was created in this animal by exposing the round window and introducing a small needle through the round window membrane into the osseous spiral lamina about 4.5 mm. from the basal end of the cochlea. Final audiograms taken one month later revealed normal thresholds for the frequency range 62.5 c.p.s. to 4,000 c.p.s. There was a threshold elevation of 40 db for 8,000 c.p.s. and no response to 16,000 c.p.s. delivered at an intensity of 55 db above the preoperative threshold (see Fig. 6).

One month after the operation the cortical test of auditory function was performed, following which the animal was sacrificed.

The thresholds for the cortical responses were essentially normal for frequencies from 125 c.p.s. to 2,000 c.p.s. There was a progressive eleva-

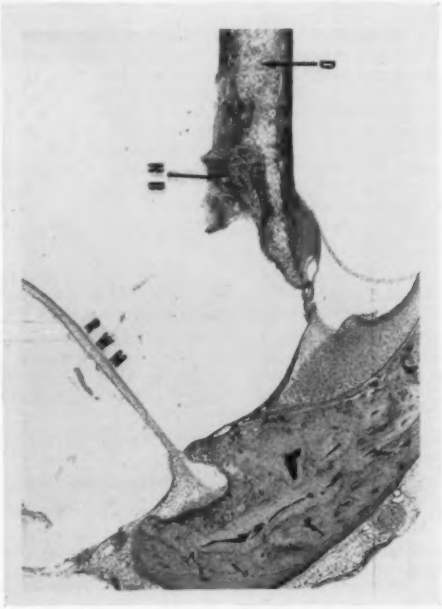

Fig. 7. From the 4.5 mm. region of the cochlea of Cat 4 showing the round window membrane (RWM), new bone formation (NB) at the point of injury to the osseous spiral lamina and the absence of spiral ganglion dendrites in the nerve canal (D).

tion of the threshold for frequencies above 2,000 c.p.s. to a maximum of 54 db for 16,000 c.p.s. The thresholds improve above 16,000 c.p.s. to become normal for frequencies of 35,000 c.p.s. to 45,000 c.p.s.

Histopathology: Histological examination revealed a lesion of the osseous spiral lamina, 3.5 to 5 mm. from the basal end of the cochlea. The inferior bony shelf of the osseous spiral lamina was fractured and partially healed by new bone and connective tissue (see Fig. 7). This scar tissue had invaded the channels of the osseous spiral lamina, which normally contain the spiral ganglion and its nerve fibres. Ganglion cell counts revealed a striking loss of these cells between the 3 and 7 mm. areas, with total loss occurring between the 4 to 5 mm. points. The spiral ganglion elsewhere was essentially normal.

The structures of the cochlear duct, including the organ of Corti and its hair cells, appeared normal. The vestibular membranous labyrinth and vestibular nerves appeared normal.

The fibres of Rasmüssen's (Olivo-cochlear) bundle were clearly visible in midmodiolar sections of the basal and middle turns and appeared to be normal.

Cat 5: An operation was performed to obliterate the cochlear aqueduct. Two and one-half months later the auditory thresholds were normal by the behavioral test. An operation for partial section of the cochlear nerve was then performed. As the thresholds were still normal three weeks later, a second more extensive cochlear nerve section was performed. On behavioral testing after the latter operation, the animal occasionally raised his ears to maximum stimulus intensities (100 db± above the preoperative threshold) for the frequency range 500 to 4,000 c.p.s., but on no occasion made the correct response of moving forward in the rotating cage. The animal was presumed to be profoundly deaf. The cortical test was done one and one-half months after the nerve section gave responses only for the frequency range 1,400 to 20,000 c.p.s. at intensities of about 60 db above average normal threshold.

Histopathology: The cochlear aqueduct was obstructed by new bone and connective tissue.

There was severe degeneration of the structures of the cochlear duct in the upper two and one-half turns of the cochlea with partial replacement of fluid spaces of the scalae with connective tissue and new bone.

The organ of Corti was present only in the basal 6 mm. of the cochlea and in this area inner hair cells were present. The spiral ganglion appeared normal in the basal 4 mm. of the cochlea. Degeneration of the ganglion was progressive from the 4 to 6 mm. area and apicalward to this region no spiral ganglion cells were seen.

The cochlear nerve trunk was degenerated except for a bundle of fibres going to the basal 4 mm. of the cochlea.

Degenerative changes with obliteration of the lumen are seen in many of the vessels within the modiolus.

Rasmüssen's bundle was missing.

Cat 6: Operations were performed to obliterate the cochlear aqueduct and endolymphatic sac. The auditory thresholds remained normal. Then a partial section of the cochlear nerve was done. One month later the auditory thresholds were still normal, so that a second nerve section was performed. Following this operation the animal gave no response to auditory stimuli of high intensity and was considered to be totally deaf. There were no responses to the cortical test three weeks after the operation, following which the animal was sacrificed.

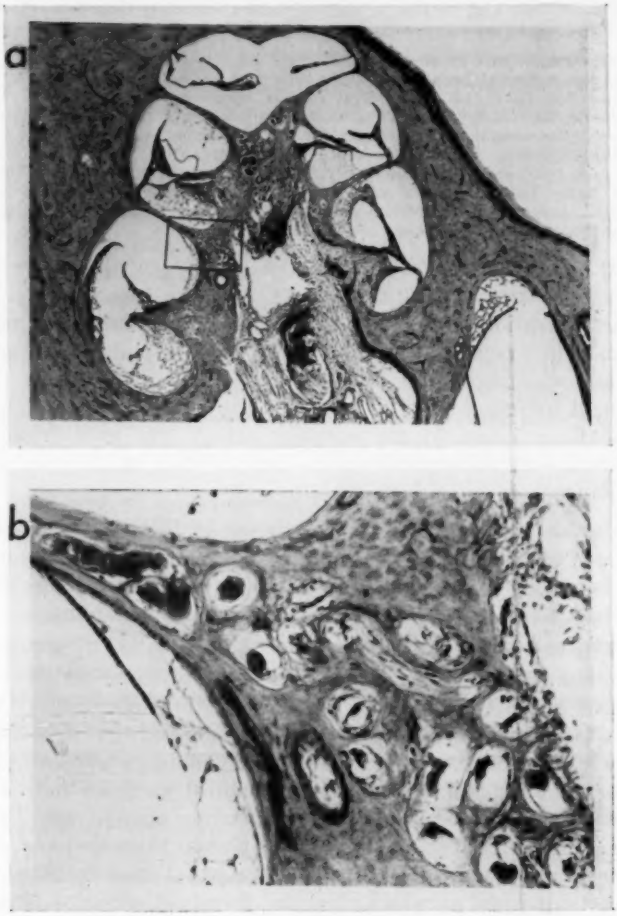

Fig. 8. (A) Cochlea of Cat 6 showing severe degenerative changes following injury to the cochlear artery (see text for description). (B) High power view of area indicated in Fig. 8 (A) to show degenerative change in vessels following injury to cochlear artery.

Histopathology: Histological study revealed that the surgical procedure had not obliterated the cochlear aqueduct; however, the endolymphatic sac was destroyed.

The cochlear and vestibular nerves were completely degenerated, and the facial nerve was partially degenerated.

The cochlea was in an advanced stage of degeneration. The organ of Corti was missing throughout. Only remnants of the basilar and tectorial membranes could be seen. The limbus could be identified, but was shrunken and distorted. The osseous spiral lamina was partially resorbed. The scalae were partially filled with connective tissue. The vestibular labyrinth was also severely degenerated with connective tissue and new bone occupying the perilymphatic spaces in some areas. There were some patent vessels with normal walls in the internal auditory meatus; however, most of the vessels within the cochlea were severely degenerated and obliterated (see Fig. 8).

Rasmüssen's bundle was missing.

COMMENTS.

An operation to cut partially the cochlear nerve was performed in five animals. In three (Cats 1, 2 and 3) the procedure was successful in that part of the nerve was cut without inflicting other injury upon the cochlea. In the other two (Cats 5 and 6) the cochlear artery was inadvertently injured, with subsequent severe degeneration of the cochlear structures.

Another animal (Cat 4) is included in this study because the ear exhibits a neural lesion of the basal coil. In this ear the injury was inflicted by introducing a needle through the round window membrane into the osseous spiral lamina.

Thus the cochleas of four of the animals (Cats 1, 2, 3 and 4) sustained purely neural lesions. Each of these ears had a normal appearing organ of Corti, including hair cells. The tectorial membrane, Reissner's membrane and the stria vascularis of each ear is normal. These findings support the generally accepted concept that the spiral ganglion may degenerate without imposing a degenerative change upon the organ of Corti. The cochlear microphonic response was recorded from the round windows of Cats 1 and 2, using a steady sinusoidal stimulus. Normal threshold stimulus intensities and normal voltage output at suprathreshold intensities were recorded in these ears. Using the click stimulus, the threshold of the microphonic response as well as the output voltage at suprathreshold intensities was normal in the ears of Cats 1 and 2. The threshold for the just visible N_1 responses to clicks was also normal in these ears. The maximum

output voltage for the N_1 response, however, was only one-fourth to one-sixth that of the normal ear. The latter finding is undoubtedly related to the diffuse severe degeneration of the spiral ganglion in these ears and supports the generally accepted view that the N_1 response to clicks represents activity in the spiral ganglion cell bodies or their fibres.

In three animals (Cats 1, 2 and 3) the central nerve fibres (axones) of the bipolar spiral ganglion cells were cut, whereas in Cat 4 the injury involved in part the peripheral nerve fibres (dendrites) of the spiral ganglion cells. Both types of injury appeared to result in irreversible and total degeneration of the involved neurones.

The results in Cats 1 and 2 were somewhat similar and can be discussed together. In both animals there was a severe hearing loss for frequencies up to 4,000 c.p.s. and near normal thresholds for the range from 8,000 c.p.s. to 25,000 c.p.s. The behavioral and cortical tests of auditory function were in fairly good agreement except at 125 c.p.s. The reason for this discrepancy is not entirely clear at this time; however, the failure of the cortical test to reveal an equally severe hearing loss may be related to the fact that relatively high sound pressure level is required at this frequency to elicit a cortical response in the normal animal. A discrepancy of the magnitude shown casts serious doubt as to the reliability of the cortical test as now performed as an indicator of the auditory threshold for frequencies below 250 c.p.s.. The behavioral test which involves a discriminatory response to sound stimulation is after all the only true test of hearing.

The behavioral test for Cat 1 was made by one of the authors (H. F. S.) and two other individuals experienced in the behavioral testing method. The audiograms made by all three operators gave thresholds within an intensity range of 5 db, except for 4,000 c.p.s., where the range was 10 db.

The ears of Cats 1 and 2 show severe degenerative changes in the spiral ganglion. In the apical one-half of the spiral ganglion of Cat 1 only 6 to 20 per cent of the cells remain. This change seems to correspond with a severe hearing loss

for the frequency range 62.5 c.p.s. to 4,000 c.p.s. In the basal one-half of the cochlear the per cent of the ganglion cells remaining varies from 20 to 40 per cent and the hearing thresholds are normal for 8,000 c.p.s. and 16,000 c.p.s. by the behavioral test and near normal from 8,000 c.p.s. to 35,000 c.p.s. by the cortical test.

The findings in Cat 2 are somewhat similar. The hearing loss for the 1,000 c.p.s. to 4,000 c.p.s. frequency range would appear to be related to the neural loss between 10 and 15 mm. where only 12 to 16 per cent of the cells remain. The threshold remains markedly depressed for the frequencies tested below 1,000 c.p.s. in spite of the presence of 35 per cent of ganglion cells between 15 and 18 mm., and 57 per cent at 19 mm. About 25 to 45 per cent of the ganglion cells remain in the region from 5 to 10 mm., corresponding to the near normal thresholds for the frequency range 8,000 c.p.s. to 16,000 by cortical test. In terms of ganglion cell density the region between 15 and 19 mm. is as well supplied as the area between 6 and 10 mm.; however, the auditory thresholds are comparatively much higher for frequencies having their regions of maximum excitation in the 15 to 19 mm. region.

The findings in Cat 3 present a significant deviation from those in Cats 1 and 2. The distribution of ganglion cell degeneration is very similar to that of Cat 2; however, the auditory thresholds were normal by behavioral test, except for a 15 db loss in sensitivity for the 4,000 c.p.s. frequency. The cortical test, on the other hand, showed a 25 to 35 db loss for frequencies up to 4,000 c.p.s. and near normal thresholds for the range 8,000 c.p.s. to 20,000 c.p.s.

The reason for the difference in threshold levels of the cortical and behavioral tests is not clear. It is possible that a sound conduction lesion was produced at the time of cortical testing, which would explain an elevation of thresholds by the cortical test.

Another possible explanation for the discrepancy between the cortical and behavioral test is that on behavioral testing, the animal was hearing with the opposite ear. This could hap-

pen if the operation to destroy the function of the opposite ear had been unsuccessful 10 months before.* One event in this animal's protocol suggests that the threshold responses by behavioral testing were not due to hearing in the opposite ear. An operation was performed through the auditory bulla. Actually this operation failed to enter the cochlear aqueduct. A behavioral test performed one week later revealed a 30 to 40 db hearing loss for all frequencies, which is a characteristic consequence of surgery in the bulla or middle ear. The thresholds improved progressively on weekly tests and were normal one month postoperatively. Such a loss would not have been evident had the auditory thresholds of the opposite ear been normal.

The histological findings do not provide an adequate explanation as to why the threshold sensitivity for low frequencies is normal for Cat 3 and markedly depressed for Cat 2. Perhaps in the ear of Cat 2 a high percentage of those fibres supplying the sensitive outer hair cells were cut, whereas in Cat 3 more of these remain.

The findings in Cats 1, 2 and 3 indicate that the threshold sensitivity for frequencies above 4,000 c.p.s. is normal with about 20 to 40 per cent of the spiral ganglion remaining in the region of the cochlea serving those frequencies. The evidence from Cat 1 suggests that a severe hearing loss for low tones can be expected when there is a loss of all but 6 to 20 per cent of the ganglion cells supplying the apical one-half of the cochlea; furthermore, the findings in Cat 3 indicate that when slightly greater amounts (up to 40 per cent) of the ganglion remains in the apical one-half of the cochlea the thresholds for low frequencies may not be affected.

In Cat 4 a discrete loss of spiral ganglion cells in a 2 mm. region of the lower basal coil has resulted in a depression of auditory threshold for the frequency range 8,000 c.p.s. to 30,000 c.p.s.

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*The opposite ear of this animal is not available for study as it was destroyed in a laboratory fire.

An examination of the audiogram and cochlear chart of Cat 4 shows that the hearing loss as well as the severe ganglion degeneration relate to the 4 to 6 mm. area as measured along the organ of Corti. This tends to support our concept of frequency localization as well as our ideas regarding the location within the cochlea of anatomically related sensory and neural units.

Thus by selective subtotal injury to the first order neuron (spiral ganglion) it has been possible to create both low and high tone hearing losses. This suggests that the auditory spectrum has a special representation in the first order neurons, such as has been demonstrated for the sense organ and for the second order neurons in the cochlear nucleus.[14]

The pathological changes which followed section of the cochlear artery are shown in Fig. 8. The spiral ganglion and the membranous labyrinth have completely degenerated, leaving only remnants of the spiral ligament, Reissner's membranes and limbus. The bone of the modiolus and osseous spiral laminae are partially resorbed. The central canal of the modiolus, Rosenthal's canal and part of the fluid spaces of the scalae contain a myxofibrous avascular connective tissue. The vessels in the internal auditory meatus have been washed free of blood by the process of arterial perfusion. The vessels in the bone of the modiolus appear shrunken in their bony channels, have thin atrophic walls and contain blood in spite of arterial perfusion, which suggests that they are functioning poorly, if at all.

DISCUSSION.

The existence of normal auditory thresholds for low frequencies in the presence of severe neural degeneration has been reported also by Neff,[5] by Wever and Neff[10] in an experiment on partial section of the auditory nerve in cats.

They cut the cochlear nerve at the internal auditory meatus. Some of their animals had severe high tone hearing losses with good hearing for low frequencies. The cochleas of these animals showed a severe degenerative change in the spiral

ganglion with almost no ganglion cells remaining in the basal portion of the cochlea and with varying amounts near the apex.

They found normal behavioral thresholds for frequencies as high as 8,000 c.p.s. with less than 10 per cent of the ganglion cells remaining in the basal 13 mm. of the cochlea. They also found normal thresholds for frequencies below 1,000 c.p.s. when only about 5 per cent of the ganglion was present in all but the apical 3 or 4 mm., where more cells remained.

The afferent nerve fibres supplying the cochlea can be classified grossly into two groups on the basis of their anatomical distribution, one group supplying mainly the inner hair cells and the other supplying the outer hair cells. The latter fibres take a spiral course for considerable distances in both the apical and basal direction in the inner and outer spiral bundles and may innervate a number of outer hair cells. The fibres to the inner hair cells take a direct route and innervate one or at most two of three cells.[11]

If these two groups of fibres have a random distribution within the nerve trunk, then cutting the nerve should involve an equal percentage of the innervation to inner and outer hair cells. If the fibres should not be randomly distributed, it would be theoretically possible to cut a higher percentage of fibres to outer hair cells in one ear than in another and still have identical ganglion cell counts. This question takes on added significance when it is realized that, given a normal nerve, the threshold sensitivity is presumably a function of the outer hair cells and their nerve fibres for stimulus intensities well above normal threshold, possibly as high as 50 db in the cat.[12]

The experimental results raise some interesting questions as to the relationship between quantity of auditory nerve fibres and auditory threshold: *1.* How many nerve fibres need be excited to create a just noticeable auditory sensation? *2.* Is this quantity the same for low tone and high tone stimulation? *3.* Is this quantity the same when excitation is occurring in fibres ending on outer hair cells as when it is occurring in fibres supplying inner hair cells in the absence of outer hair

cells? *4.* Is the quantity of nerve fibres required to create a just noticeable auditory sensation a function of the density of innervation? For example, if a threshold response can be elicited by exciting a certain number of fibres ending on a small area of the sense organ, can this same physiological effect be achieved by exciting this same number of fibres distributed over a larger area?

Another consideration relates to the fact that the resonance curves on the cochlear partition (at suprathreshold intensities) are much broader for low frequencies. Does this favor neural excitation by low frequencies in the nerve-injured ear?

Obviously the experimental evidence does not provide the answer to these questions. From the results of Cats 1 and 2 and 3 it seems justifiable to state that: *1.* the auditory thresholds may be normal for frequencies above 4,000 c.p.s. when there remains only 20 to 40 per cent of the spiral ganglion cells serving those frequencies. *2.* The auditory thresholds for the frequencies 4,000 c.p.s. and below are markedly depressed when there remains only 6 to 21 per cent of the ganglion cells serving those frequencies, but may be normal or depressed when 20 to 50 per cent of the ganglion cells remain in the apical region of the cochlea.

It is interesting to note that the saccular macula and saccular nerve were intact in the ear of Cat 1, in which ear there was a marked threshold elevation for low frequencies. Thus it appears that the saccule does not serve as a receptor for low frequency stimuli.

It should be understood that ganglion cell density (expressed in terms of the per cent of cells remaining) may not exist as a direct function of threshold intensity for all frequencies of the auditory spectrum. It may be that in the middle region of the cochlea (upper basal coil), where the absolute number of ganglion cells per unit length of organ of Corti is highest, the greatest percentage of cells must be lost to cause a threshold elevation. This might be true if the just noticeable auditory sensation is dependent upon the activation of a uniform minimum number of nerve fibres at all frequen-

cies. If this is true, the ganglion cell degeneration in our animals would relate more directly to threshold if expressed in terms of the actual number of ganglion cells remaining per unit length of the organ of Corti.

Such a method was used by Guild *et al.*,[13] who made a correlation of ganglion cell degeneration and changes in auditory threshold for human ears. Their method was to make ganglion cell counts from every fifth section throughout the cochlea and to determine from these values the number of spiral ganglion cells per half-turn of the cochlea. As the evaluations are for very long regions of the organ of Corti, the method has the disadvantage of failing to demonstrate changes of limited size and severity. They demonstrated a correlation between threshold elevations for high frequencies and atrophic changes in the spiral ganglion of the lower part of the basal turn of the cochlea.

The concept that large numbers of cochlear nerve fibres can be lost without a demonstrable change in the threshold audiogram is not new. This was demonstrated by Dandy after cochlear nerve section of patients with Ménière's disease and by Neff in a study of experimental cochlear nerve section in animals. It is logical that a small number of nerve fibres are adequate to conduct an impulse of threshold magnitude. On the other hand, such an ear would be expected to perform poorly for psychoacoustic phenomena requiring large numbers of nerve fibres.

For example, large numbers of cochlear nerve fibres are probably required for normal pitch and loudness discrimination. Defects in ability to discriminate pitch and loudness should manifest themselves in the form of poor speech discrimination scores in human patients. Thus, it would seem theoretically possible to have normal auditory thresholds, but diminished speech discrimination in an ear with subtotal nerve degeneration. Observations made on a recent patient support this thesis. A 31-year-old woman with an acoustic

neurinoma of the left ear was found to have very poor speech discrimination in that ear in spite of normal auditory thresholds for frequencies in the speech range 512 c.p.s. to 4,096 c.p.s., inclusive.*

CONCLUSIONS

1. Subtotal degeneration of the spiral ganglion occurred in four cats without a concurrent morphological change in the organ of Corit. In these ears the microphonic response to steady sinusoidal stimuli and clicks was normal. The threshold for N_1 response to clicks was also normal; however, the maximum output voltage for the N_1 response to clicks was diminished.

2. By selective section of cochlear nerve fibres restricted hearing losses were produced for both low tones and high tones. These findings support the concept that there is spatial representation in the spiral ganglion for low tones as well as high tones.

3. The evidence indicates that the threshold for any frequency of the auditory spectrum remains normal when 20 to 40 per cent of the spiral ganglion cells remain in the region of the cochlea serving the frequency, but that a threshold elevation is likely to exist when less than 20 per cent of the ganglion cells remain.

4. Diminished speech discrimination appears to be an earlier sign of degenerative change in the spiral ganglion than elevation of the auditory threshold.

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*This case will be reported in detail in another publication.

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CARDIAC ARREST: ITS ETIOLOGY AND TREATMENT.*

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(by invitation)

Cardiac standstill may occur at any time before, during or after anesthesia and surgery, and regardless of when it does occur its effect is terrifying to the surgeon, and its results are sometimes demoralizing and devastating to the operating team. This increasingly occurring complication is now common newspaper story material, but fortunately, or unfortunately, these cases reported in the news items are the occasional patient who survived this dreaded complication, whereas many more cases of cardiac arrest occur every day and do not survive. Many of you may never experience having a patient develop a cardiac arrest, and I hope that none of you shall have such a misfortune; nevertheless, all of us must know precisely what to do in the event that we are confronted with such a situation.

First of all, it would be well to discuss certain aspects and factors in the cardiac resuscitation itself, and most important obviously are the etiological factors in cardiac arrest during anesthesia and surgery. These are an overdose of the anesthetic agent, overdose of other medication, reflexogenic basis (vagovagal reflex), which is an alteration of the normal conduction mechanism, the diseased myocardium, whether it be due to acute myocardial failure or inadequate coronary circulation, and finally, hypoxia of the myocardium itself, due either to moderate respiratory obstruction or hypotension. Other factors are the differentiation of cardiac standstill from ventricular fibrillation, which is most essential. In cardiac standstill the heart is motionless, and progressive cyanosis ensues; in ventricular fibrillation, one sees that the heart looks

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like a bag of worms and ineffective waves of contraction spread over the ventricles. Finally, an important factor in cardiac resuscitation is the restoration of function, which is possible providing proper steps are taken at the proper time. Obviously, immediate cardiac resuscitation is mandatory for successful recovery.

There are certain factors in the cardiorespiratory failure which are most important to discuss briefly, or at least to enumerate. Death is due to interruption or alteration of respiration or circulation, and the danger signals are evidenced by a chain of circumstances which leads to a breakdown in the effective oxygen delivery system to the heart and to the brain. Resuscitation, then, consists of two main components, first, the delivery of oxygen to the lungs, and second, the circulation of oxygen through the blood stream by an effective and efficient heart. Oxygen may be delivered to the lungs either by mouth to mouth technique employed until a bag or a mask can become available, and then the most effective method of oxygen delivery is by squeezing a rubber bag filled with oxygen and forcing it through an intratracheal tube to the lungs. Adequate ventilation of the lungs with oxygen having been established, and compression of the heart instituted, the crisis is over, even though spontaneity of beating and respiration has not returned. Most important of all, for complete recovery of the patient, the oxygen delivery and circulation must be accomplished within a few minutes after the oxygen system has broken down.

I shall mention only briefly certain physiological factors of cardiac resuscitation. The fundamental physiological properties of the heart are that it possesses excitability, rhythmicity, conductivity, and contractility. The speed of conduction within specialized neuromuscular portions of the heart varies, and the fibres in the A. V. node have a conduction rate of 200 mm. per second, the ventricle conduction rate is 400 mm. per second, that of the auricle is 800 mm. per second, and the fibres of Purkinje have a conduction rate of 4,000 mm. per second. The refractory period of the heart is relatively long and serves to preserve the cardiac rhythm. Vagal stimulation

shortens the refractory period of the heart muscle, whereas certain drugs, such as quinidine and procaine, prolong the refractory period. The heart that has gone into standstill due to vagovagal reflexogenic reflexes usually returns to normal more easily than a heart that has gone into standstill due to blood loss or anoxia; furthermore, the diseased myocardium invariably takes longer to return to spontaneous heart beat after it has gone into arrest. The usual causes of ventricular fibrillation are anoxia, mechanical trauma and coronary insufficiency (coronary occlusion). It is possible to produce ventricular fibrillation by mild electrical shocks of 0.09 to 0.4 amperes current, whereas a current much stronger, 1.5 to 3.8 amperes of current, defibrillates in the heart.

Certain pharmacological aspects in the treatment of cardiac arrest are most important to know. When the heart stops, the ineffective circulation may be due to cardiac standstill or ventricular fibrillation, and the therapy for restoration to normal beat is different in each of these two conditions. An electrocardiogram may be helpful in differentiating between the two, but they can be clinically recognized; the electrocardiogram is not necessary, and usually it is not available at the time that cardiac arrest takes place. It is known that the calcium ion increases the contractility and prolongs systole, whereas the potassium ion reduces contractility and prolongs diastole. Finally, the rôle of the sodium ion is not exactly known, but excitability and contractility are not maintained in its absence, experimentally. The rôle of the autonomic nervous system, also, is important to recognize. The parasympathetic nervous system is stimulated by such drugs as acetylcholine, mecholyl, and prostigmin, whereas its inhibitors are atropine and scopolamine. The sympathetic nervous system, on the other hand, is stimulated by epinephrine, norepinephrine, isopropylnorepinephrine, neosynephrine, and methoxamine, whereas the inhibitors of the sympathetic division are ergotamine and dibenamine. There are certain cardiac drugs that are used as stimulants, such as digitalis, ouabain, and nikethamide, and cardiac depressants, such as quinidine, procaine and procaine amide (pronestyl). The central

nervous system drugs that are sometimes employed are metrazol, picrotoxin, amphetamine, strychnine, all of which are stimulants. The depressants of the central nervous system are morphine and barbiturates.

In cardiac arrest where the patient has obviously no circulation, the left ventricle is the most effective site for introduction of the drug and initiation of the circulation of the drug into the coronary circulation and thereby supplying the myocardium. The right auricle is the second best site of injection, and certainly drugs should not be used until the heart is first rhythmically compressed, for it is not helpful to introduce the drugs into the peripheral veins until adequate circulation is established. The routine intravenous administration of drugs is of little or no value in cardiac arrest and is to be condemned; however, rapid intravenous fluid and blood may greatly aid in carrying medication to the heart, and to maintain adequate fluid volume cardiac output during manual compression of the heart. Epinephrine and Norepinephrine are pre-fibrillatory agents, especially after standstill with anoxia and with anesthetic agents such as chloroform and cyclopropane. Drugs, plus the rhythmical manual compression in most normal hearts give co-ordinated heart beats.

The treatment of cardiac standstill then, comprises manual compression of the heart with injection of epinephrine, 0.2 of a milligram (0.2 cc. of a 1:1,000 solution) injected into the left ventricle. Continued manual compression is necessary for at least thirty minutes unless the heart begins to beat spontaneously before that time. Procaine, 50 to 100 milligrams (5.0 to 10.0 cc. of a 1% solution) may be used to counteract fibrillation of the ventricle if necessary. Epinephrine and isopropylnorepinephrine increase cardiac rhythmicity, contractility and excitability; therefore, these drugs predispose to ventricular fibrillation, and for that reason one should limit the amount of epinephrine used. Neosynephrine and methoxamine, on the other hand, are not cardiac stimulants and do not predispose to ventricular fibrillation. One of the most valuable drugs in the treatment of cardiac arrest is calcium chloride, 2.0 to 4.0 cc. of a 10% solution injected into the left ventricle,

which in itself may bring recovery when all other agents have failed. It is our practice then, to introduce calcium chloride immediately into the ventricles of our patients who have cardiac arrest. The use of barium chloride is not effective.

The treatment of ventricular fibrillation is indeed different. Obviously, manual compression of the heart is kept up until an effective spontaneous beat is obtained, but in this condition, procaine, 50 to 100 milligrams (5.0 to 10 cc. of 1% solution) into the left ventricle is a cardiac depressant; it prolongs the refractory period, decreases the conductivity and the excitability. If one should be fortunate enough to get a standstill after procaine, one should administer epinephrine, 0.2 cc. of a 1:1000 solution. Usually, however, application of the electric shock in serial defibrillation is necessary and if the conditions are proper, the muscle fibers contract vigorously with the electrical shock and when the shock is broken, the myocardium remains completely relaxed. Thus, one gets a temporary standstill. One may use procaine, 9.5 cc. of a 1% solution, and epinephrine, 0.2 cc. of a 1:1,000 solution immediately thereafter, and continue the manual compression. When the spontaneous beat returns, it feels almost as though the heart is jumping out of the hand.

The surgical approach to the heart, and the effective procedure of resuscitation of the heart are essential factors for the success of cardiac resuscitation and the recovery of the patient. The time element is all important, and all that is necessary is a knife and a pair of hands, for it must be remembered that the patient with cardiac arrest is virtually dead: there is no circulation and, therefore there is no bleeding. It must be mentioned that one should not waste time to get a stethoscope or to determine whether the heart is stopped, when the indications are apparent. Compression under the diaphragm is usually ineffective and the injection of epinephrine through the chest wall is likewise of no value in the vast majority of cases. If one waits long enough for an electrocardiogram and wastes time in dilating the rectal sphincter and attempts to give mechanical respiration by chest compression one is wasting valuable time. If there is no palpable pulse, no

blood pressure, respiration has ceased, then any slight heart action is of no consequence and will probably be ineffective. It is necessary then, to open the chest wall immediately, for delay in the resuscitation of the heart by manual compression is usually the cause of failure, in most instances.

The procedure for cardiac resuscitation is more or less an orderly one. One must open the chest through the fourth or fifth intercostal space on the left without wasting time for asepsis. The heart can be exposed quickly in a matter of thirty to sixty seconds in order to begin manual compression rhythmically, with the fingers being compressed sixty to eighty times a minute. The insertion of a tracheal tube in order to deliver oxygen to the lungs is most important and should be done within the first three to five minutes. After the incision has been enlarged, the patient's heart can be grasped and compressed rhythmically while rib spreaders are being placed for more adequate exposure. The rhythmic compression of the heart must be continued at all times except for the few seconds that are necessary for the injection of drugs into the left ventricle. If it so happens that the right side of the chest is already open, then the right sided approach to the heart is quite adequate. Usually, the forceful administration of oxygen by mask to the patient dilates the stomach with oxygen, and the insertion of a Levine tube into the stomach should be carried out later on, for a distended stomach is not conducive to good cardiorespiratory function. One should continue with rhythmic compression of the heart for at least thirty minutes before abandoning this procedure. The effectiveness of the compression of the heart may be determined by a radial pulse or a carotid pulse. If spontaneous beating of the heart occurs with an absent blood pressure, then the compression should be continued in order to deliver an effective volume of blood to the circulation, until adequate blood pressure returns. If spontaneous beating occurs with a blood pressure of systolic of fifty or more, then one of the pressor agents, such as neosynephrine or methoxamine may be given in small doses. When a weak, ineffective beat does return, it is well to attempt to conform to rhythmic compression by reinforcement of the spontaneous beat itself, rather than attempting to compress

the heart more frequently than it wishes itself to beat. The carotid blood flow and the cerebral blood flow is obviously enhanced by the occlusion of the aorta, which may be clamped off temporarily in patients who have no blood pressure. Where ventricular fibrillation occurs, one should continue with manual compression of the heart and artifical respiration with 100% oxygen until the defibrillation apparatus is available. If, after ten to twenty minutes, no blood pressure is obtainable with no cardiac response to compression or medication, there is little hope for successful resuscitation. Once compression and medication have effected a return of cardiac activity, one cannot assume that cardiac automaticity is completely restored. Vigorous contraction of the heart under influence of drug therapy may cease after the peak of the drug stimulation has passed. One cannot predict recurrence of arrest in the subsequent twelve to twenty-four hours.

It is recommended that a drug tray be set up in the operating room in case of emergency, and this drug tray should contain the following drugs:

1. Epinephrine—0.3 cc. of 1:1,000 solution
2. Isopropylnorepinephrine—0.02 mgm.
   (Isuprel, Aleudrine, Norisidrine)
3. Calcium chloride—10% solution
4. Atropine—1/50th grain
5. Procaine hydrochloride—1% solution
6. Normal saline—100 cc.

In addition, a sterile instrument package containing a defibrillator apparatus and the following instruments should be kept sterile in the operating room for an emergency cardiac arrest:

1. Rib Spreader (1)
2. Hemostats (3)
3. Medicine Glasses—1 oz. (4)
4. Scalpel (1)
5. Scissors (1)
6. Forceps (2)
7. Syringes, 10 cc. (4)
8. Sponges

Since time is of the essence, it is obvious that the anesthetist and the surgeon have certain duties, and these duties must be carried out with dispatch and no waste of time. The anesthetist should notify the surgeon immediately when the pulse and blood pressure disappear, and the surgeon may check the absence of pulse by palpation of the aorta or heart if he is at the time working in the chest, or to check the cartoid pulses or the other major arteries by palpation. The anesthetist should immediately insufflate the lungs with 100% oxygen by a breathing bag and mask, and when feasible, introduce an intratracheal tube for more efficient oxygenation of the lungs. He should likewise count the time from the onset when he recognized the absence of pulse and blood pressure for the record.

Briefly then, in review, the surgeon in the instance of a cardiac arrest should direct his team to carry out immediately a thoracotomy incision over the fourth or fifth left interspace in order that he may carry out cardiac manual compression. Epinephrine, 0.3 cc. of a 1:1,000 solution diluted in 10 cc. of normal saline should be introduced directly into the left ventricle, and if not, into the right ventricle. Atropine, grains 1/50 in 10 cc. of normal saline solution may be introduced into the left ventricle. Calcium chloride, 2 to 4 cc. of 10% solution, diluted in 10 cc. of normal saline solution directly into the left ventricle. If, upon examining the heart it is seen that ventricular fibrillation is present, then cardiac compression must be continued throughout until the heart is restored to normal spontaneous beat. Here procaine hydrochloride, 5 or 10 cc. of a 1% solution, is introduced directly into the left or the right ventricular cavity, and electric shock directly to the heart of at least 2.5 amperes for one full second. If this fails to arrest the fibrillation, then increased amperage and increased time of the shock may eliminate the fibrillation. If asystole occurs after defibrillation or after procaine, then epinephrine, 0.3 cc. of a 1:1,000 solution diluted in 10 cc. of normal saline may be necessary to introduce into the left ventricular cavity. The patient should be kept in Trendelenburg position and the electrocardiogram may be of help following

the various administration of drugs and cardiac compression; however, cardiac compression should not await electrocardiogram. Cautious use of the pressor drugs, intravenous fluids, and blood after the restoration of the heart beat are obvious. One must not over treat the patient medically, and one must not increase the blood volume circulation unduly. One should observe the heart for twenty to thirty minutes in the operating room before closure of the chest. In all cases where the chest has been opened for cardiac compression, the pleural cavity should be drained by an intercostal catheter brought out through a stab wound, and judicious use of the antibiotics is likewise indicated.

BILATERAL PARALYSIS OF THE VOCAL CORDS: TREATMENT BY TRANSORAL ARYTENOIDECTOMY.*†

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The surgeon whose patient has paralysis of both vocal cords following thyroidectomy may suffer unjust criticism or worse. Those to whom such patients come later for treatment may be asked to explain the reason for the problem, and unless careful consideration is given to the reply, such criticism may be increased rather than allayed. It should be remembered that one of the recognized hazards of thyroidectomy is the possibility that a recurrent laryngeal nerve may be injured and cause paralysis of the corresponding vocal cord. Because of certain variable factors, such as anomalous situation of the recurrent laryngeal nerve or its displacement by tumor, this complication is encountered in a small percentage of cases following thyroidectomy regardless of the skill of the surgeon and in spite of his utmost care to avoid it. A frank discussion along these lines should enable the patient to understand that no one was at fault.

SYMPTOMS AND SIGNS.

It has been my good fortune to have had the opportunity to examine the larynx in a large number of patients before and after thyroidectomy. Some 25 years ago, over a period of approximately three years, it was my duty in connection with an active surgical service to make preoperative examinations of the vocal cords of all patients who were to undergo thyroidectomy and 10 to 14 days later to make corresponding postoperative examinations of the same patients. These ex-

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aminations were made by indirect laryngoscopy. If a postoperative examination revealed that the patient had suffered paralysis of a vocal cord, inquiry was made regarding any untoward symptoms he may have experienced during the early postoperative days. Careful records of the replies were kept until a series of 88 such cases had been accumulated. For comparison, corresponding records for patients who had not suffered vocal cord paralysis were kept. When 1,300 such records were available, this control series was terminated. The results of these observations are shown in Table 1.

TABLE 1. HOARSENESS OR CHOKING OR BOTH DURING THE FIRST 10 DAYS AFTER THYROIDECTOMY.

Patients with	Hoarseness, Per Cent	Choking, Per Cent	Both, Per Cent
No vocal cord paralysis	3.4	2.4	0.2
One vocal cord paralyzed	95.5	81.9	80

The important point to note in Table 1 is that 80 per cent of the patients who have suffered paralysis of one vocal cord have had both hoarseness and choking during the first few postoperative days, whereas the combination of these two symptoms was noted in only 0.2 per cent of those patients who had no such paralysis. It can be said, therefore, that if a patient has both hoarseness and choking during the early postoperative period he probably has experienced paralysis of at least one vocal cord.

In the course of this study it was observed that occasionally the postoperative examination would reveal a paralyzed vocal cord in a patient who had not the usual early symptoms. Some of these patients had noted slight hoarseness beginning after several postoperative days, and the question arose as to whether a vocal cord might become paralyzed several days after operation. It was decided, therefore, to make an examination of the larynx of each thyroidectomized patient within a few hours after operation in addition to the usual examination made 10 to 14 days later. This study revealed that in an occasional patient the larynx would be normal immediately after operation but a paralyzed cord would be observed 10 to

14 days later. On re-examination of such patients several months later, it was observed that the paralysis had disappeared. I can offer no explanation for the occurrence of this late paralysis, but have wondered whether it might be due to edema within the sheath of the recurrent laryngeal nerve.

One of the surgeons whose patients were under observation during this study declined to accept the idea that late paralysis of the vocal cords could occur until after a most convincing incident that involved one of his patients. The examination made immediately after operation had revealed no paralysis. The patient had an absolutely uneventful convalescence and at noon on the ninth postoperative day the surgeon had told her she might leave the hospital. Three hours later, tracheotomy became necessary owing to bilateral paralysis of the vocal cords. Examination several weeks later revealed that both vocal cords had regained normal function. I have seen no other case of late bilateral paralysis.

A knowledge of the behavior of a vocal cord after it has been paralyzed during thyroidectomy makes it easy to understand the ensuing symptoms. When a vocal cord thus has become paralyzed it, of course, becomes immobile* and almost invariably, at first, its medial margin is bowed outward. Gradually and over a length of time which varies from a few days to six months this outward bowing disappears, and the margin of the vocal cord again becomes a straight line. The explanation for this gradual change is not known. The position which this immobile cord eventually assumes is at or near the position a normal vocal cord assumes during phonation or, in other words, at or near the median line of the larynx. Thus in the case of a patient who has suffered paralysis of only one vocal cord two things are noteworthy six months or more afterward: the first is that the paralyzed cord is in good position for phonation so that when its normal partner moves to a corresponding position the conditions for speech are essentially normal, hence the patient is not hoarse. The second is that the patient practically never complains of dysp-

*For all practical purposes the paralyzed vocal cord is immobile, but in a considerable percentage of cases laryngoscopic examination reveals that it does move slightly. It is not understood why this is true in some patients.

nea. This is because the normal airway is about twice as large as needed for normal activities and because the paralyzed vocal cord obstructs not more than one half (usually somewhat less than half) of the airway; therefore, since such a patient usually has neither hoarseness nor dyspnea, the condition often will be unrecognized by his physician, and the patient rarely will be aware that there has been any change in his vocal and respiratory apparatus.

Since paralysis of one vocal cord so easily may escape detection unless laryngoscopic examination is made and since it does reduce the airway to one half of normal, it must be remembered that if this patient's other vocal cord should become paralyzed his half-capacity airway will undergo considerable further reduction and he then may have only one-fourth the normal capacity or even less. Such narrowing of the airway, if it occurs suddenly, necessitates immediate tracheotomy. This emphasizes the fact that if a patient is to have a second thyroidectomy it is very important that the larynx be examined before operation so that the surgeon may be forewarned and forearmed if the patient already has one paralyzed vocal cord.

I have a suspicion that some respiratory emergencies during thyroidectomy which have been attributed to tracheal collapse have instead been caused by paralysis of a vocal cord in a patient whose other vocal cord previously had been paralyzed.

Perhaps I should digress here to point out the entirely different behavior of a paralyzed vocal cord when the lesion producing the paralysis is not that of surgical trauma. In such cases the paralyzed cord assumes a position usually well lateral to the median line of the larynx, its margin is bowed outward, and it remains bowed. (The patient, of course, will be hoarse.) This fact has an important practical application. If the laryngologist examines a previously thyroidectomized patient and finds a paralyzed vocal cord in a lateral position and bowed, he can say with confidence, "Do not attribute this

paralysis to the thyroidectomy — rule out carcinoma of the thyroid or esophagus, a lesion in the mediastinum, at the jugular foramen or elsewhere."

In the course of numerous preoperative laryngeal examinations I have observed what I have chosen to call inhibitory fixation of a vocal cord. Only rarely is it observed. At first on the examination of a patient who has had no operation on the neck, one vocal cord may be seen to remain immobile at the median line of the larynx. The experienced examiner will say to himself: "This patient has no history to suggest any possibility of surgical trauma and if this immobile vocal cord has become paralyzed owing to some other lesion it should not lie at the median line of the larynx." He will realize that it probably would be an error to report "paralysis of one vocal cord."

In cases in which this has been observed the patient usually has been very nervous and tense. If the examiner is suspicious, he should simply tell the patient that he will return in half an hour to look at her throat again. Knowing what the second examination will be like, she probably will be relaxed and usually it will be observed that both vocal cords move normally. Rarely is it necessary to anesthetize the palate.

Now let us apply the known facts regarding the behavior of the vocal cord paralyzed by surgical trauma to the course of events following the occurrence of simultaneous surgical paralysis of both vocal cords. Both vocal cords would be immobile and at first both would be bowed; therefore, during the early postoperative period the patient would be very hoarse or even aphonic. There would be no dyspnea because of the ample airway between the bowed vocal cords; also, in the early postoperative period the patient would be likely to have considerable trouble with choking because the glottis cannot be closed to keep liquids from entering the trachea. Because of this, the patient might suffer from aspiration pneumonia, and the first few days of the postoperative course probably would be very stormy. I never have had an opportunity to observe, during the immediate postoperative period, a patient who had suffered simultaneous surgical paralysis of

both vocal cords. I have talked with numerous patients, however, who have had this disability, and their descriptions indicate that their symptoms have corresponded closely with those to be expected and as outlined.

During a period of four to six months after the onset of bilateral paralysis of the vocal cords the bowing of the cords gradually changes so that the margins become more nearly straight. As this change occurs, the hoarseness improves because the vocal cords are approaching nearer and nearer to the normal position assumed during phonation. Usually when the change is complete the voice is practically normal. During the same period the patient becomes increasingly dyspneic because the above described change in the vocal cords causes gradual reduction in the airway. The final degree of dyspnea will depend upon how near to the median line of the larynx the paralyzed cords take their position. This varies with different patients, and it is not known why there is this variation. In some patients the space between the cords is wide enough to provide as much as 30 to 40 per cent of the normal airway, and the patient may be able to carry on quite comfortably. In most instances, however, the airway is reduced to 25 per cent of normal or less and the patient is severely handicapped with respect to physical activities. The narrowing of the airway takes place so very gradually that some patients adjust themselves so well that they live in reasonable comfort with an airway that is not more than 20 per cent of normal.

Patients who have bilateral paralysis of the vocal cords react badly to opiates and to barbiturates, and they tolerate general anesthesia very poorly.

DIAGNOSIS.

The diagnosis of post-thyroidectomy simultaneous bilateral paralysis of the vocal cords readily can be made simply by talking with the patient if the condition is suspected and has existed six months or more. The patient's chief complaint will be that of shortness of breath on exertion. Appropriate questioning will elicit the history of thyroidectomy, followed immediately by loss of voice, by several days of acute illness,

and later by gradual improvement in the voice, accompanied by the appearance of gradually increasing dyspnea.* It will be noted that inspiration is prolonged and accompanied by a low musical tone in the throat. If the physician next will ask, "Do your relatives say that you make a lot of noise when you sleep?" the reply will be, "Oh, yes—it is terrible—I even waken myself." This symptom, "terrible noise while sleeping," almost can be said to be pathognomonic. It is a real problem both for the patient and for his relatives. One patient's primary complaint was "I want to travel but I cannot because I disturb other people so much when I sleep."

TREATMENT.

Treatment for bilateral paralysis of the vocal cord is indicated whenever the degree of dyspnea is such that the patient cannot live comfortably and happily within the limitations imposed by it.

Some patients have separation of the paralyzed cords sufficient to provide an airway which permits them to live comfortably with moderate restriction of physical activities, and treatment should not be urged for them. It should be borne in mind, however, that such patients may need treatment later in life when age reduces the margin of physical reserve so that they no longer can live comfortably with their handicap. I have had two such patients who came for treatment at the age of 60 after having had the problem for 19 years, and a third who came at the age of 62 after 20 years.

Other patients may be so severely handicapped within six months or a year after the onset of the paralysis that surgical treatment should be advised. I believe, however, that no patient should be operated upon earlier than six months after the onset because occasionally late recovery of vocal cord function does appear.

Patients with bilateral paralysis of the vocal cords not uncommonly suffer from thyroid and parathyroid insufficiency.

*In the case of a patient who suffers paralysis of one vocal cord at one operation and of the other at a later one, the dyspnea usually appears immediately after the second operation. The subsequent history will be similar, however, and the ultimate symptoms the same.

Before surgical treatment is undertaken, search should be made for evidence of such complications, and suitable treatment should be instituted if any deficiency is found. One of my medical colleagues, Dr. L. P. Howell, has given me the following statement regarding such investigations and treatment:

"As a rule, the diagnosis of myxedema can be made on the basis of the history, physical examination and basal metabolic rate. Determination of blood cholesterol may be helpful. If the diagnosis of myxedema is doubtful, collateral information can be obtained by an estimation of the serum protein-bound iodine and an estimation of thyroid function by means of tracer studies with radioiodine. Parathyroid insufficiency should be suspected when there is a history of paresthesias and tetany. A positive Chvostek sign is suggestive; a positive Trousseau sign is indicative. The presence of incontrovertible hypocalcemia and hyperphosphatemia is pathognomonic.

"Myxedema can be controlled readily by the use of desiccated thyroid gland. The management of postoperative parathyroid insufficiency is tedious and sometimes difficult. If hypocalcemia is not marked, the oral ingestion of cacium lactate may suffice. As a rule, however, the ingestion of calcium salts must be supplemented with either vitamin D or, preferably, dihydrotachysterol. Periodic, and sometimes frequent, determinations of blood calcium are necessary to make sure the patient remains in metabolic balance."

At the present time it is rather generally agreed that the choice of treatment rests between two methods. One is a method whereby the arytenoid is removed or is anchored laterally with the field of operation exposed through an external incision. This method, of which there are several modifications, often is referred to as the King[1] operation because Dr. King was the first to develop the plan for it. The other is a method whereby the arytenoid is removed with the field of operation exposed by means of the suspension laryngoscope. This method is referred to as the Thornell[2] operation, for Dr. Thornell, who first described it. Those surgeons who have not been trained in the use of the suspension laryn-

goscope will prefer the King type of operation. Those who have had such training will prefer the Thornell operation. Good results can be achieved by either method and I can see no need for any quarrel between the advocates of the respective methods. The surgeon simply should select the method he best knows how to use.

Upon learning of Dr. Thornell's operation, my laryngologic colleagues and I immediately were favorably impressed because we long had used the suspension laryngoscope in the treatment of patients who had tumors of the larynx. We now have employed his method in the surgical treatment of 50 patients who had suffered bilateral paralysis of the vocal cords (see Table 2). In one of these patients the paralysis was due to acromegaly.

TABLE 2. BILATERAL PARALYSIS OF THE VOCAL CORDS: TREATMENT BY TRANSORAL ARYTENOIDECTOMY (50 PATIENTS).

Results	Patients	Remarks
Excellent	43	Five required operation on both sides
Fair	2	One happy with elimination of noisy sleeping — needs operation on other side One writes, "better but not enough" — needs operation on other side
Unsatisfactory	1	Previously had King operation on both sides—slight improvement
Too recent for evaluation	4	Three should have good result One may need operation on other side

SUMMARY.

If, during the first five postoperative days, a thyroidectomized patient is hoarse and also suffers from choking, it is highly probable that at least one vocal cord has been paralyzed. Six months or more after one vocal cord has been paralyzed at thyroidectomy the patient probably will have no symptoms referable to such paralysis. Six months or more after both vocal cords have been paralyzed at thyroidectomy the patient's voice usually will be essentially normal but he will have more or less dyspnea. Treatment for bilateral

paralysis of the vocal cords is indicated whenever the patient cannot live comfortably within the limitations imposed by his dyspnea.

ADDENDUM

Since this paper was written, the 4 patients whose results are listed in table 2 as "too recent for evaluation" have been decannulated and can now be classified as having "excellent" results.

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THE USE OF APOLAMINE AS A PREOPERATIVE ANTINAUSEA AGENT FOR LARYNGOSCOPY, BRONCHOSCOPY AND ESOPHAGOSCOPY.

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Endoscopic procedures performed for the examination of the air and food passages may be unnecessarily uncomfortable for the patient and somewhat unsatisfactory for the examiner if the patient is nauseated, retching or vomiting. Factors producing nausea and vomiting are extremely varied, and thus no specific single agent could be considered as effective routinely or even in similar pathologic conditions for which the examinations are being made. For these reasons, a polypharmaceutical agent, Apolamine®,‡ has been used as a preliminary medication for laryngoscopy, bronchoscopy and esophagoscopy in adult patients to be examined under local anesthesia. This study has been based upon records of nausea or vomiting preceding or following endoscopy with and without the use of this agent. This preliminary report seems warranted to indicate that the results are favorable enough to justify a thorough, controlled series.

DESCRIPTION AND ACTION.

Each Apolamine tablet contains atropine sulfate 0.1 mg., scopolamine hydrobromide 0.2 mg., phenobarbital 15 mg., benzocaine 100 mg., pyridoxine 2.5 mg., nicotinamide 25 mg. and riboflavin 4 mg.

The composition of Apolamine is designed to counteract various inciting factors and physiologic accompaniments of

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nausea and vomiting. Phenobarbital reduces excitability of the vomiting center by its central sedative effect. This action is reinforced by the presence of scopolamine, which also acts with atropine in decreasing gastric secretion, thereby allaying nausea. Benzocaine produces moderate topical analgesia in the stomach, minimizing the tendency for emesis initiated by local reflex irritation. The three members of the vitamin B Complex group are useful in that pyridoxine helps to overcome nausea and vomiting in pregnancy; nicotinamide diminishes gastrointestinal motility; and riboflavin deficiency has been correlated with vomiting during pregnancy.

CLINICAL RESULTS.

This agent has been used in 1,154 cases as a preliminary medication for laryngoscopy, bronchoscopy and esophagoscopy. It has augmented the routine preoperative sedation of morphine, atropine and sodium phenobarbital that had been employed in the majority of cases since the phenobarbital and atropine content was not considered sufficient for the desired protective effect of the barbiturate against untoward action of the local anesthetic nor the desired secretion reducing effect of atropine. Under other conditions for which Apolamine is used, the dosage of these two agents would be considered sufficient.

Local anesthesia used in the endoscopic procedures consisted of a pharyngeal spray of 2 per cent pontocaine followed in five minutes by careful drop-by-drop instillation of 2 cc. of 10 per cent cocaine onto the posterior surface of the epiglottis, the vocal cords and into the trachea.

USAGE.

The occurrence of nausea and vomiting before, during or after peroral endoscopic procedures using the preoperative medication and local anesthesia mentioned above, with the exception of Apolamine, is approximately 11 per cent, a figure gained from a review of 2,912 adult cases in the two years prior to the use of Apolamine. With the addition of Apolamine in the series of 1,154 cases, there were 48 patients, or

4 per cent, who vomited or complained of nausea before, during or after the endoscopic procedure.

There has been no occurrence noted of any undesirable side action.

DISCUSSION.

Apolamine has been studied in a series of 135 cases of nausea and vomiting in early pregnancy.[1] Many of these cases involved women who had vomited so much during this and previous pregnancies that they were unable to do their housework. In 102 cases (73.3 per cent) of this series, Apolamine efficiently controlled the nausea and vomiting. It was observed that in some patients, after 10 days of treatment, the vomiting did not recur when treatments ceased. The dose ranged from one to three tablets daily.

The efficacy of Apolamine tablets was evaluated in a series of 755 cases of nausea and vomiting involving a wide variety of clinical conditions.[2] The average dose was three tablets taken daily with meals. In the 135 cases in this series treated for nausea and vomiting of pregnancy one to two tablets were administered daily for from six weeks to four months.

One is skeptical of any polypharmaceutical agent of this type since there are a multiple of possible pharmacological actions; however, the clinical impression of the action of this agent has been favorable, although records based upon patient-reaction are admittedly subject to possible error and the studies were not controlled except with comparison to previous case records of the same group performed in the same manner. This preliminary report is warranted to indicate that the results are favorable enough to justify a more controlled series under rigid investigative conditions. This preliminary report also justifies the continued clinical use of the drug.

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ANHYDROUS FURACIN EAR SOLUTION IN THE TREATMENT OF CHRONIC SUPPURATIVE OTITIS MEDIA WITH OBSERVATIONS ON THE USE OF TRICHLORACETIC ACID IN THE CLOSURE OF TYMPANIC MEMBRANE PERFORATIONS.*

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Kinston, N. Car.

This study was initiated in February, 1948, to determine the efficacy of 5-nitro-2-furaldehyde semicarbazone(furacin) in the treatment of chronic suppurative otitis media. The anhydrous solution containing 0.2 per cent furacin in a polyethylene glycol base was employed throughout. All of the patients treated were seen in an office practice that is predominantly private in nature. As was to be expected, certain difficulties were encountered in securing all desired studies and cooperation in treatment of some of the patients.

PLAN OF PROCEDURE.

The general plan of procedure consisted in eliciting an adequate history and making a thorough examination of the ears, nose and throat, including visualization of the nasopharynx and transillumination of the antra and frontal sinuses. Examination of the tympanic membranes was made by reflected light supplemented by magnification. The Eustachian tubes were routinely inflated by catheter method.

Pretreatment hearing tests included whispered voice tests, two audiograms, and Rinne, Weber and Gelle tests. Audiometric tests were made on two successive days from 8:00 to 9:00 A.M. in a small inner office room lined with acoustic celotex. The author made all hearing tests with the same model 50-E-ADC audiometer, which was calibrated at 18-

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month intervals. Masking was used when indicated. The Rinne test was made with magnesium alloy tuning forks from 512 through 4,096 d.v., inclusive. The Weber test was made at the vertex of the skull, glabella, upper central incisor teeth and chin. All hearing tests were repeated in the same manner at the conclusion of treatment of those patients who secured a dry ear and healed perforation. Other auxiliary studies were made as often as possible or when warranted.

INCIDENCE.

The incidence of chronic suppurative otitis media in the general population of this country is variously estimated at 1 to 4 per cent. The official census for the nation in 1950 revealed that there were 150,697,361 persons within the continental limits of the United States.[1] This indicates that there are approximately 1,506,973 to 6,027,894 cases of chronic suppurative otitis media in this country at the present time. Records compiled by the national headquarters of the Selective Service System show that the incidence of otitis media per 1,000 registrants examined at local boards from April, 1942, through December, 1943, was 15.6 for whites, 2.3 for Negroes and 13.6 for all races.[2] While not specifically stated, it is assumed that at least the majority of these cases represented the chronic form of suppurative otitis media. Myersburg[3] found 30 cases of chronic suppurative otitis media out of 400 patients seen in the office practice of otolaryngology.

In my study a total of 4,784 new patients were seen in office practice from Feb. 1, 1948, through Dec. 31, 1951. In this number there were 145 cases of chronic suppurative otitis media, including eight chronic draining radical mastoid cavities. This gives an incidence of 3 per cent.

In this total number of 145 patients the right ear was draining in 56 cases, the left ear in 67 cases, and both ears in 22 cases. There was, therefore, a total of 167 chronic discharging ears. The shortest duration of otorrhea was one month and the longest was 44 years. The series included 81 males and 64 females; 142 whites and three Negroes. The age distribution of these patients is recorded in Table 1.

Only 55, or 37.9 percent, of the 145 patients reported regularly for treatment. Of this number, 25 were males and 30 females.

TABLE 1. AGE DISTRIBUTION OF 145 CASES OF CHRONIC SUPPURATIVE OTITIS MEDIA.

Years	Number of Patients	Per Cent
1-10	28	19.3
11-20	34	23.5
21-30	23	16.0
31-40	20	14.0
41-50	27	18.6
51-60	5	3.5
61-70	5	3.5
71-80	1	0.7
Not recorded	2	1.3

FACTORS RESPONSIBLE FOR CHRONICITY OF AURAL DISCHARGE.

Opinion varies as to when an acute otitis media becomes chronic. Older concepts considered that an ear which had been discharging for two months might be thought of in terms of chronicity. Kopetzky[4] and Shambaugh,[5] however, pointed out that the underlying pathology in any given case, and not the duration of otorrhea, should be the vital criterion for determining chronicity.

Some of the factors responsible for chronic suppurative otitis media are known. Local conditions in the nose, throat and oral cavity generally acknowledged to contribute to the chronic running ear are well known and need no particular review. Cullom[6-8] stressed the relationship of chronic suppurative sinusitis in certain cases of chronic otorrhea. Only two cases of such nature were noted in this series of patients.

Some cases of chronic running ears are presumably due to a chronic infection in the Eustachian tube. There is relatively little information extant in the literature regarding anatomical variations or pathological conditions of the Eustachian tube and their influence in promoting chronic infection in this region. The anatomy of this structure is known to vary with the age of the patient and in different individuals. Spielberg[9] demonstrated that the normal course of the Eustachian tube is that of an inverted "S." Corrugations of the tubal mucosa

and variations in vascularity and amount of cilia present were described by Polvogt and Babb.[10] Variation in the shape of the tubal lumen, accessory cartilages and cartilaginous fissures were noted by Graves and Edwards.[11] Welin[12] demonstrated abnormal course and irregularity of contour of the tube, local and diffuse strictures, polyp-like structures and peritubal air cells. Altmann[13] described developmental anomalies of this area, such as congenital diverticulae, polyps and hyperplasias. Bast and Forester,[14] Wolfe,[15] Crowe,[16] Lindsay[17] and Unger[18] made contributions to the knowledge of peritubal air cells, but there is a difference of opinion among some of these authors regarding their significance in promoting chronic infection in the tubal region.

Farrior[19] studied the effect on the tubal mucosa of various kinds of infection, allergy and trauma from improper adenoidectomy, repeated catheterization and manual dilatation. He described the microscopic changes which take place as a result of these conditions and concluded that they tend to produce mechanical obstruction and promote chronic infection by interference with normal tubal function.

Many cases of chronic suppurative otitis media develop as a result of repeated acute infections of the middle ear structures, especially if untreated or poorly managed. Predisposing factors to these infections include any of the local conditions already mentioned in addition to well known environmental and diseased states which affect the general health adversely. Acute infections which develop in a middle ear with a hyperplastic or fibrous mucosa (Wittmaack), a constitutionally weak mucosa (Albrecht) or an allergic mucosa (Dohlman) may be destined to chronicity because of the limited resistance to infection and the poor capacity of this type of mucosa to heal. A brief but lucid account of these theories was presented by Koch.[20] Sellers,[21] Tumarkin[22] and others expressed the opinion that inadequate chemotherapy and biotherapy in the treatment of acute suppurative otitis media may lead to immunity of the pathogenic organisms, latent infection, reduced pace of the pathologic process, and eventually a chronic infection, especially in the attic region.

Shambaugh[5] stated that most cases of chronic running ears develop as a result of acute necrotic otitis media, a peculiar condition seen in infections of high virulence among young children with lowered resistance. It is prone to develop in some cases of scarlet fever, measles and influenza.

Cholesteatoma (epidermoid cyst,[23] inclusion cyst,[24] cholesteatosis[25]) is a frequent cause of chronic suppurative otitis media. The neoplasm, immigration and metaplasia theories have been advanced to explain the histogenesis of cholesteatoma. The pros and cons of these various theories have been ably discussed in the literature and recently by Begley *et al.*[26] These authors demonstrated prickle cells in the lining of an aural cholesteatoma, thereby establishing this layer as being truly epidermic in nature. Attic suppuration is intimately associated with the presence of cholesteatoma in many cases, but not all. McGuckin,[27] after his clinical study of attic disease stated that it may be insidious in its development and already an established chronic disease when first manifest. On the other hand, his observations led him to conclude that attic disease may proceed to complete resolution without subjective complaint in some cases.

While trauma in warfare is a fairly common cause of chronic suppurative otitis media from blast injury, barotrauma or foreign bodies, it is not a frequent cause of this condition in civilian practice. The problem of foreign bodies in the external auditory canal as a cause of this condition was ably discussed by Simonton.[28] Foreign bodies in the Eustachian tube are a very rare cause of chronic suppurative otitis media.[29]

Chronic running ears of tuberculous or syphilitic nature are not commonly seen in office practice. According to Myerson,[30] the incidence of tuberculous otitis media is higher in young children where the infection supervenes by way of the blood stream in most instances. Shea[31] stated that mycotic infections of the middle ear and mastoid are more common than the literature indicates.

An analysis of the 145 cases of chronic suppurative otitis media under consideration revealed that the majority were attributed to recurrent acute abscesses in one or both ears. In most cases there was a history of repeated myringotomies. In some instances the cause of the otorrhea was not known to the patient. In 25 patients a diagnosis of secondary acquired cholesteatoma was reasonably certain. Three patients attributed their chronic running ears to measles, two to swimming, three to whooping cough, one to whooping cough and pneumonia, one to typhoid fever and pneumonia, one to pneumonia, two to scarlet fever, and six to trauma. In the traumatic group there was one case each due to auto accident, blast injury, slap on the ear, injury in football, removal of foreign body from the ear, and scalding water. One case of chronic otorrhea in a child 12 months of age was proven to be of tuberculous origin.

SYMPTOMS.

The symptoms of uncomplicated chronic suppurative otitis media depend largely upon the nature and location of the pathologic process and duration of the disease. The two most characteristic symptoms are aural discharge and impaired hearing.

The discharge may be continuous or intermittent, mucoid or purulent, odorless or foul, scant or profuse, or blood streaked. In cases of bone necrosis without cholesteatoma there is usually a foul continuous discharge. The drainage when cholesteatoma is present, while occasionally profuse, is characteristically scant in proportion to the lesion, foul, and often contains flakes of cholesteatomatous material. In the so-called "tubal ear" the drainage is usually mucoid or mucopurulent, odorless, intermittent and varies in amount. In addition malodorous aural discharge may result from stagnation, "saprophytic infection," fungus and fusospirochetal infections, and occasionally from an associated chronic suppurative sinusitis. Blood streaking or bleeding is usually associated with the presence of granulations and occasionally malignancy.

In the majority of patients in this series the aural discharge was mucopurulent, intermittent and varied in amount. Many patients with this type of drainage frequently noted the presence of a foul odor, but it seems likely that in most cases the odor could be attributed to stagnation from neglect, obstruction or inability to thoroughly cleanse the ear at home. Twelve patients gave a history of frequent blood streaking, and in three of this group profuse bleeding from the ear was the primary cause for the patients' seeking medical attention. In these latter cases the source of bleeding was a large mass of granulations which filled the external auditory canal.

The hearing loss in chronic suppurative otitis media varies. Hughson[32] stated that clinical and experimental data show that the conductive mechanism of the ear contributes 40 to 50 per cent of the total normal hearing acuity, possibly less but of itself never more. Asherson[33] conducted an audiometric investigation on 500 consecutive cases of chronic otorrhea in World War II and found a profound and permanent loss of hearing in more than 90 per cent of cases. In 153 consecutive cases of chronic otorrhea with granulations present there was an average hearing loss of over 40 db for all frequencies and for 39 cases of aural polypi an average hearing loss of 45 db. Guild[34] stated that impairment of thresholds by air conduction may be approximately equal for all tones, greater for low tones than for high, or greater for high tones than for low tones. Maxwell[35] summarized the problem of hearing loss in chronic suppurative otitis media by stating that it is largely influenced by the amount of damage done to the middle and internal ear at the onset of the acute infection, extent of bone necrosis and degree of interference with the bone conduction mechanism during the process of healing. Thus there may be present a conductive type of deafness of mild or severe degree, a mixed deafness if cochlea damage be present, or in some instances a severe nerve deafness.

In this series complete hearing tests were made on 74 patients with 88 chronic running ears, excluding the eight patients with draining radical mastoid cavities. There was a mixed deafness of varying degree in 77 ears, nerve deafness

in six ears, and normal hearing in three ears. One additional patient with extensive bilateral cholesteatoma of the middle ear and mastoid and bilateral and postauricular fistulas presented a total loss of hearing in both ears. Of the remaining 63 patients with 71 draining ears, the hearing for whispered voice was not recorded in 11 ears, was absent in 14 ears, and was normal in 19 ears. In 16 ears whispered voice was heard at a distance of between one and five feet, in four ears between six and 10 feet, and in seven ears between 11 and 14 feet.

Persistent pain of appreciable degree is not a characteristic symptom of chronic suppurative otitis media and when present suggests that tension has developed from obstruction to drainage or that a complication of more serious import has developed. The intractable pain present in the patient with bilateral cholesteatoma was completely relieved by bilateral radical mastoidectomy. Tinnitus when present is usually of mild degree but may be troublesome. The majority of patients in this series had an associated external otitis due in part to climate, irritating effect of the aural discharge, and the trauma of cleansing. In some instances repeated acute exacerbations were troublesome and associated with a recurrence or increased amount of aural discharge.

SIGNS.

a. Perforations. The tympanic membrane in cases of chronic otorrhea is generally much thickened and retracted and often adherent to the promontory. The perforation is usually single, but in syphilitic, tuberculous and mycotic infections multiple perforations may be present. There are two general types of perforations, the central and the marginal. The former does not involve the periphery of the pars tensa; varies in size, form and location; is rarely associated with bone necrosis; and usually represents a nondangerous type of otitis media. It may occur with or without granulation tissue or polyp formation and is commonly seen as a result of obstruction to the ventilation and drainage of the Eustachian tube. The marginal perforation shows destruction of the periphery of the drum head and annulus tympanicus and represents a poten-

tially dangerous form of aural suppuration, since there may be caries of the adjacent bone. Like the central type, it may occur in variable size, form and location. The most common type of marginal perforation occurs in the posterior-superior quadrant of the membrana tensa and adjacent Shrapnell's membrane.

An especially dangerous type of perforation occurs in the membrana flaccida and is commonly referred to as an attic perforation. A perforation in this region may be small, covered by an adherent crust and difficult of demonsration. It may indicate suppuration of the attic and tympanic antrum, caries of the incus and the possibility of cholesteatoma formation. In this connection the observations of Tumarkin[22] are of interest. He stated that attic inflammation may be acute or chronic and that in each case an "enclosed" stage, difficult of clinical diagnosis, results before overt discharge and perforation appear. He believes that "enclosed epitympanitis" in the pneumatized mastoid is due to inadequate antibiotic therapy and therefore responsible for some cases of obscure deafness. Finally, he pointed out that cholesteatoma of the epitympanum produces an "anemic necrosis" of the incus because of its normally poor blood supply. The insidious development of chronic "enclosed epitympanitis" explains the insidious development of marginal perforations in this area. Total loss of the tympanic membrane is not common, since the annulus tendinosus is somewhat resistant to suppurative disintegration.

CLASSIFICATION OF TYMPANIC MEMBRANE PERFORATIONS IN PRESENT STUDY.

For the purpose of classification and discussion in the present study a perforation of the membrana tensa has been classified as anterior, inferior or posterior central, according as it occurred in front of, below or behind the handle of the malleus without concomitant involvement of the annulus. In like manner a perforation of Shrapnell's membrane has been designated as anterior, central or posterior attic, according to its relation to the head of the malleus. Marginal perforations have been classified in the conventional manner. A perfora-

tion with the major portion of the defect in the membrana tensa without involvement of the annulus, but with extension into the adjacent Shrapnell's membrane has been classified as an anterior or posterior-superior perforation. This classification of perforations is represented diagrammatically in Fig. 1.

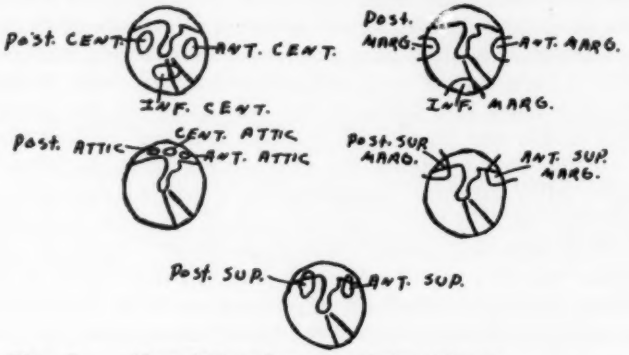

Fig. 1 -- Classification of perforations. (Diagrammatic).

In the present series of 159 chronic running ears, eight radical mastoid cavities excepted, the nature of the perforation was not determined in two ears and not recorded in four ears. The remaining 153 ears presented 154 perforations. These were classified as anterior central in 16 ears, inferior central in 33 ears, and posterior central in 33 ears. In seven ears there was a posterior-superior perforation. Seventeen ears had a posterior-superior marginal perforation and one ear an anterior-superior marginal perforation. Eight ears presented a posterior marginal and two ears an inferior marginal perforation. There were 11 attic perforations: five anterior attic, four central attic and two posterior attic. A rim of tympanic membrane remained in 16 ears, and there was a total absence of the tympanic membrane in 10 ears. The ear with a double perforation presented a small anterior attic and large posterior central defect. One of these ears with a posterior central defect was proven at operation to have an extensive cholesteatoma of the middle ear and mastoid.

b. Granulations and Polypi. Granulation tissue and aural polypi when seen in conjunction with chronic otorrhea are evidence of chronicity. They may arise from any denuded surface of the middle ear or external auditory canal and appear as single or occasionally multiple growths of varying size, consistency and color. They may be attached by a broad base or narrow pedicle. Granulation tissue may simply reflect an attempt at healing of a superficial ulceration in the presence of infection or may indicate underlying bone disease. In some cases this tissue is the sole cause of the chronic otitis media, and its removal may effect a cure. Where there is an underlying osteitis the granulation tissue persistently recurs after removal. Occasionally, repeated removal may minimize the infection with limitation of necrosis, absorption of dead bone and cure.[36] Wilkerson *et al.*[37] reported good results with the use of radium therapy in the control of recurrent aural granulations. Ten patients were treated by introducing a 50 mgm. nasopharyngeal radium applicator into the middle ear and maintaining it in place for 12 minutes. Radium therapy was instituted on the third to sixth day after surgical removal of the granulations.

Lederer[38] pointed out that certain polyps in the external auditory canal often seen in conjunction with chronic otorrhea may represent such neoplastic formations as carcinoma; specific granulomas such as may be associated with tuberculosis and syphilis; or manifestations of certain metabolic diseases. Excluding these types, he classified aural polyps into four major groups, including the granulation polyp, the connective tissue polyp, the granulation polyp acutely exacerbated, and the lymphatic polyp. This classification was confirmed by Wilkerson *et al.*[37] with the addition of fibroma as a separate entity.

In this study visible granulations or polypi were present in 56 ears for an incidence of 33.5 per cent. Granulations were removed and pathological studies made in 22 patients. In six specimens the granulations were covered in whole or in part by squamous epithelium and in two specimens by squamous epithelium and columnar ciliated epithelium. One speci-

men was lined by pseudostratified columnar ciliated epithelium, and in the remaining 13 specimens no epithelium was present. In these 22 sections an acute inflammatory reaction was present in five. In five sections there was a profusion of eosinophiles and in two a profusion of multinucleated giant cells. Two specimens revealed cystic degeneration and one specimen extensive hemorrhage. In one specimen removed from the ear of a child with pulmonary tuberculosis there was typical tubercle formation.

AIDS IN DIAGNOSIS AND EVALUATION OF CHRONIC SUPPURATIVE OTITIS MEDIA.

Laboratory studies are of some value in the diagnosis and evaluation of any given case of chronic suppurative otitis media and include cultures, cytology, studies for cholesterol crystals and mastoid X-rays.

Interest in the bacteriology of chronic running ears has been revived since the introduction of the sulfonamides and antibiotics into clinical otology, and sensitivity tests have enjoyed a more widespread use. Changes in the bacteriological flora of chronic suppurative otitis media treated by local application of antibiotics and sulfonamides have been repeatedly demonstrated. Hayes and Hall[39] advanced the interesting concept that there is a seasonal variation in the flora of otogenic infections, the Gram-positive organisms flourishing in the Winter months and the Gram-negative organisms prevailing in the Summer. Most cases of chronic otorrhea are due to a mixed infection with staphylococci, streptococci, diphtheroids, B. pyocyaneus and Proteus vulgaris. Fungi, fusospirochetes and other organisms may be present in some cases. Bacillus pyocyaneus and Proteus vulgaris, formerly considered in the light of harmless saprophytes, are now considered by many to possess definite pathogenic properties. Out of 100 cases cultured by Fowler[24] only 26 cases showed a pure growth, a finding in keeping with the majority reports on the bacteriology of chronic aural suppuration. The identification of fungi by smear and culture in cases of mycotic infection of the middle ear is difficult, as is the identification of the tubercle bacillus in cases of aural tuberculosis.[31,30]

Cytologic studies of aural secretions are of relatively recent innovation. They are made to show the type, number and condition of the leucocytes; the presence or absence of tumor cells, epithelial cell forms and fatty epithelial degeneration; the number of bacteria; and the amount of mucus present. Some efforts along the line of cytologic studies of aural discharge were made by Shih-Ping Ts'en,[40] Proetz,[41] Dean and Pfingsten[42] and Hansel.[43]. Dohlman,[44] however, was apparently the first to undertake a large scale cytologic examination of the discharge in various types of middle ear infection. He was particularly interested in the demonstration of eosinophiles in the aural discharge and the allergic investigation of these patients. Out of 178 cases he found a secretion eosinophilia exceeding 9 per cent in 99 cases and by correlated studies was able to establish a relationship to allergy. Koch[20] made cytologic studies on 222 patients with 262 chronic discharging ears. Of this number, a diagnostic amount of secretion eosinophilia was found on one or more examinations in 41 patients with 52 chronic running ears. It is of interest that in these cases there was a central perforation of the tympanic membrane in 26, a marginal perforation in two, and radical mastoidectomy had been performed in 14. Cope[45] stressed the value of cytologic studies in cases of chronic otorrhea resistant to conventional therapy. Senturia *et al.*[46] used this method to aid in the differential diagnosis of chronic external otitis and chronic suppurative otitis media. House[47] and Friedmann[48] utilized the Papanicolaou stain to study aural discharge for the presence of tumor cells.

Cytologic studies were made from 11 selected ears in the present group of patients, and in two ears there was a diagnostic amount of eosinophiles in the aural discharge.

The microscopic demonstration of cholesterol crystals plus fatty degenerated epithelium in the secretion of chronic running ears has been considered as diagnostic of cholesteatoma. The detection of cholesterol in the aural discharge by chemical analysis has been thought of as a diagnostic point for the presence of cholesteatoma, and it has been reported that the normal cholesterol index of the blood rises in cases of exten-

sive cholesteatoma of the middle ear. On the other hand, Dean and Pfingsten,[42] in their microscopic study of over 100 cases of acute and chronic abscesses of the middle ear, demonstrated that cholesterol was usually found in cases of chronic otorrhea in which cholesteatoma was not present, and when cholesteatoma was proven the discharge did not always contain cholesterol. They also showed that normal cerumen contains more amorphous and crystalline cholesterol than does cholesteatoma and that cholesterol may be present in other acute and chronic diseased conditions of the ears, nose and throat. The chemical test which they employed to demonstrate cholesterol in the aural secretion was not satisfactory for an accurate diagnosis of cholesteatoma. Lederer[49] stated that the presence of cholesterin in cholesteatoma of the middle ear is purely accidental and owes its existence to the decomposition of organic matter in an atmosphere lacking in oxygen.

In this series smears for cholesterol crystals were made from the aural discharge of 22 patients with 23 pathologic ears. In six ears cholesterol crystals were present in the discharge, but a clinical diagnosis of cholesteatoma was not made. In eight ears there was no clinical evidence of cholesteatoma, and no cholesterol crystals were found in the drainage. In six ears a diagnosis of cholesteatoma was certain, and smears were negative for the presence of cholesterol crytals. This included three ears in which extensive cholesteatoma of the middle ear and mastoid were proven at operation. In three ears the presence of cholesterol crystals in the smears was consistent with a clinical diagnosis of cholesteatoma.

X-rays of the middle ear and mastoid in cases of chronic suppurative otitis media present certain well known difficulties of technique and interpretation that have been presented in the recent literature by Fletcher and Windholz.[50] The Law and Stenver positions were used routinely in the X-ray study of the patients in this series.

Mastoid X-rays were secured in 80 patients in this group, and in all there were present varying degrees of sclerosis. No case of chronic suppurative otitis media was observed with normal pneumatization of the mastoid process. In the patient

with a central perforation of the tympanic membrane X-rays revealed an extensive cholesteatoma of the middle ear and mastoid which was unsuspected from the clinical examination.

NEW MEDICAMENTS FOR TOPICAL APPLICATION IN THE TREATMENT OF CHRONIC SUPPURATIVE OTITIS MEDIA

In order to aid in the evaluation of results obtained with anhydrous furacin ear solution the literature on the conservative treatment of chronic suppurative otitis media was reviewed from 1937 through 1951 with major emphasis on certain new medicaments introduced during this period for the treatment of this condition. These medicaments can be classified as digestant (urea, enzymol, carica papaya), chemotherapeutic (sulfonamides, dibromosalicylaldehyde, glycerite of hydrogen peroxide, furacin), and antibiotic agents (penicillin, streptomycin, tyrothricin, acetic acid, chlorophyll, palutin, homosulphamin). With two exceptions the papers reviewed were those which dealt with the topical application of these drugs without concomitant systemic administration. These agents were employed by various otologists as powders, solutions and suspensions and were introduced into the middle ear by drops, insufflation, displacement method or via Eustachian catheter. Most authors stressed the value of local cleanliness, preliminary bacteriological studies, and sensitivity tests. A striking fact brought to light in this review is that few authors mentioned the ultimate fate of the tympanic membrane perforation. Since the results obtained with these new drugs are now well known, a detailed analysis of this review has been omitted with the exception of furacin. The bibliography, however, has been included.[51-114]

FURACIN.

The furans are chemicals obtained for the most part from oat hulls. Furfural, the fundamental member of the furan series for the synthesis of other derivatives, results from the dehydration of sugars of five carbon atoms known as pentoses which are present in the natural product.[115] Furacin solution, a nitrofuran derivative, has been prepared for aural use in a watery and anhydrous form. The latter contains 0.2 per cent

furacin in polyethylene glycol 300, and it has been accepted by the Council on Pharmacy and Chemistry of the American Medical Association.[116] Shipley and Dodd,[117] the Council on Pharmacy and Chemistry,[118] Anderson and Steele[119] and Douglass[120] found the solution to be effective against most of the organisms encountered in chronic aural infections. Douglass stated that the solution is not active against fungi and that Proteus vulgaris is acted upon by furacin but requires higher concentrations than those needed to treat ears infected by other organisms. McLaurin[83] confirmed the observations of Anderson and Steele and of Douglass regarding the efficacy of furacin in coagulase positive staphylococcic aural infections. This was proven experimentally by Cramer and Dodd.[121]

Meleney *et al.*[122] and Dodd *et al.*[123] reported that the use of nitrofurazone solution does not inhibit the healing of clinical and experimental wounds. Hanzlik *et al.*[124] and Dodd[123] reported that neither the drug nor its vehicle is irritating on local application. McCullough[125] and Anderson and Steele[119] found that sensitivity to its local use in infected wounds is rare. Douglass[120] observed local reactions to furacin therapy in about 4 per cent of 86 patients treated for chronic aural infections, and he stated that this incidence corresponds to that reported by Shipley and Dodd[117] and Downing[115] in the use of furacin for treatment of skin infections. Morin[126] reported a single case of severe local and systemic reactions from the use of soluble furacin dressing on a leg ulcer, and McLaurin[83] alluded to local reactions from furacin therapy in the treatment of aural infections.

Cramer and Dodd[121] and Asnis and Gots[127] as a result of their laboratory experiments accumulated evidence which led to the conclusion that furacin acts by prolongation of the lag phase of bacterial growth due to an interference with the enzyme system of bacterial respiration. During this period the bacteria are stationary and the body defenses are in a better position to cope with them.

OBJECTIVES AND PRINCIPLES IN THE TREATMENT OF CHRONIC SUPPURATIVE OTITIS MEDIA.

The ultimate objectives in the conservative treatment of chronic suppurative otitis media are to secure a dry ear, close

the tymanic membrane perforation when feasible, and improve the hearing. In most instances the indications for conservative or surgical therapy are clearcut. In some cases, however, the decision as to which method to employ may be a difficult one to make.

The cardinal principles of local therapy in the treatment of chronic suppurative otitis media are scrupulous cleansing of the ear, often difficult of achievement, and the addition of an antibacterial agent. The opinion has been advanced that medicaments introduced into the external auditory canal rarely penetrate the middle ear system because of the complicated anatomy of this region. The intricacies of the tympanic and paratympanic cellular system demonstrated by anatomical and histological studies lend credence to this view. On the other hand, the clinical studies of Welin[12] and others indicate that solutions introduced into the external auditory canal or Eustachian tube by injection may penetrate the cellular system to a greater extent than one might reasonably expect.

The theoretical cardinal principles of local chemotherapy of chronic running ears are well known. Routine bacteriological identification of organisms and determination of their sensitivity to any given therapeutic agent in office practice has not been practical in my experience because of economic reasons and lack of local facilities for the proper conduction of this work. It is known that drugs will act on organisms in clinical infections that they have little effect on in the test tube. It seems logical that these tests might be reserved for those cases which do not show a satisfactory response after a reasonable period of treatment. That good results and clinical cures may be secured without them is evident in the forthcoming analysis of results.

CLOSURE OF TYMPANIC MEMBRANE PERFORATIONS.

Attempts to close tympanic membrane perforations have been made since the earliest days of otology. It seems that this is a vital step in the treatment of cases of chronic otorrhea when the nature of the perforation is such that it lends itself to closure. It is the unfortunate experience of every otologist to obtain a dry ear without spontaneous or induced

healing of the perforation and have the otorrhea recur with the next or subsequent respiratory infections. A healed tympanic membrane, even though scarred, is an additional safeguard for a permanently dry ear.

Although Politzer[128] showed that cicatrization of the perforated drum membrane may take place from the mucosal layer, it has been established that proliferation from the epidermal layer is more rapid. Stinson[129] studied the healing process of the tympanic membrane by clinical means and formulated certain laws of epithelial proliferation. These observations were confirmed by Lawson[68] and partly by Magnoni.[130]

Dunlap and Schuknecht[131] stated that the process of drum membrane repair stops when the stratified squamous epithelium on the external surface grows over the edges of the perforation and meets the pseudostratified columnar or cuboidal epithelium on the inner surface. Destruction of the perforation margin removes this epithelial barrier and incites by tissue destruction an inflammatory healing response in the margin of the tympanic membrane. Fibroblastic proliferation in the middle layer of the granulating margin is responsible for the laying down of interlacing fibres of collagen. The epithelium grows over the perforation edges in four to seven days and must be repeatedly destroyed. When collagen fibres are completely formed they shorten. The shrinking of this circular band of interwoven fibres may facilitate closure by purse string action.

Dunlap and Schuknecht considered the following conditions to contraindicate closure of the perforated tympanic membrane: *1.* any form of mastoid disease; *2.* presence or suspicion of cholesteatoma; *3.* destruction of the ossicular chain in some cases; *4.* continuously discharging ear (the intermittent discharging ear may be closed, but a dry period must be secured); *5.* complete loss of the pars tensa; *6.* large marginal perforations; and *7.* obstruction to the Eustachian tube.

Efforts to close tympanic membrane perforations have centered around the application of a cauterizing agent, insertion

of a prosthesis, or both. Okuneff, in 1895, is generally credited with being the first to use trichloracetic acid as a cauterizing agent for this purpose. Dunlap,[132] Linn,[133] Dunlap and Schuknecht,[131] Szpunar[134] and Adams[135] used various strengths of trichloracetic acid solution with good results. Stinson[136] and Fox[137] used Cargile's membrane successfully as a prosthesis after preliminary chemical cauterization of the perforation edges. Shambaugh[5] preferred cigarette paper for this purpose, and Steinmann[138] advocated the use of cellophane. Henry[139] used cellophane softened in alcohol as a patch to stimulate healing of dry perforations of the tympanic membrane secondary to wartime blast injuries. Hoople[140] also found the judicious use of a prosthesis or splint a valuable adjunct in the treatment of wartime traumatic perforations of the tympanic membrane. Unger[141] obtained good results with the use of scarlet red ointment and a gold foil patch.

PLAN OF TREATMENT IN THIS STUDY.

The plan of treatment used in the present series consisted in having the patient cleanse the involved ear at home with dry wipes, suction with medicine dropper, or both. Following this, he lay on the sound side with the infected ear uppermost. This ear canal was then partially filled with furacin solution and the position maintained for 15 minutes by the clock. During this time intermittent tragal pressure was instituted. At the conclusion of this period a cotton tampon was inserted tightly in the ear canal and allowed to remain in place for at least an hour. Treatment was carried out three times daily and continued for two weeks after the ear became dry. The principle of continuous therapy with furacin rather than switching from one agent to another was followed throughout in most instances. The patient was warned to discontinue treatment if symptoms of sensitivity developed.

Treatment in the office was carried out at approximately weekly intervals. As a preliminary measure large granulations were removed by snare when present. Small visible granulations were treated by application of trichloracetic acid coincident with attempts to close the tympanic membrane perforation. The latter method consisted in cleansing the

involved ear by suction and cotton-tipped applicators moistened with hydrogen peroxide or ether. Particular attention was paid to removing the dried exudate from the margin of the perforation at each visit. When allowed to accumulate, the dried exudate hindered healing and simulated a rapidly healing perforation. When excessive in amount, as on occasions when the patient failed to return regularly for treatment, the collar of exudate was removed with a dull curette after preliminary softening with peroxide. After thorough cleansing the ear was dried by compressed air. The edges of the perforation and surrounding tympanic membrane for a millimeter or so were touched with a cotton-tipped applicator moistened with a 50 per cent solution of trichloracetic acid, care being taken to avoid the skin of the external auditory canal. No anesthetic was used. Patients usually complained of pain for a short while at the first few treatments but rarely thereafter. The margin of the healing perforation presented a reddish appearance due to the presence of granulations, and when touched with the acid it immediately blanched. If blanching did not occur in a particular segment, it was again cleansed and the acid reapplied. Even with infinite care it was difficult to keep from applying small amounts of the acid to the middle ear mucosa during the process of treatment, but no untoward effects were noted except for some temporary increase in discomfort. In one case after furacin failure trichloracetic acid was deliberately applied to the mucosa overlying the promontory with a dry ear resulting.

There was no attempt to select patients for treatment with the exception of three patients (four ears) who presented definite danger symptoms and on whom a radical mastoidectomy was performed. In three cases the tonsils and adenoids were removed after a dry ear and healed perforation were secured. In one child tonsillectomy and adenoidectomy and submucous resection of the nasal septum were done after furacin failure. The surgery also failed to secure a dry ear. No septal or other form of sinus surgery was performed on any other patient.

RESULTS OF TREATMENT IN THE PRESENT SERIES.

a. Discharging Ears. The 55 patients who reported regularly for treatment have been divided into three major groups. An additional group of four patients with unilateral dry middle ears and perforated tympanic membranes has been included. The data concerning these patients has been summarized in Tables 2 and 3.

Group 1 consists of those patients who obtained a dry middle ear and healed perforation. There were 34 patients in this group with 40 chronic discharging ears. The condition was bilateral in four patients, and in two ears there was a recurrence of the otitis after the perforation was closed. In both instances a dry ear was secured a second time.

In this group 30 dry ears were secured with the initial course of furacin. This total includes two ears in which sensitivity to furacin developed, but the discharge was so diminished by this time that a dry ear and healed perforation eventually resulted. Three ears required a second course of furacin for recurrence of the otitis before the perforation was closed, and two ears required a second course for recurrence after the perforation was closed. This gives a total of 35 out of 40 ears which became dry with the initial or second course of furacin therapy. In the remaining five ears a dry state was secured by the local use of glycerite of hydrogen peroxide in one ear; by the conservative treatment of an associated chronic suppurative sinusitis in two patients; and by the use of peroral terramycin in two patients.

TABLE 2. DATA ON 55 PATIENTS WITH 65 CHRONIC DISCHARGING EARS.

Group No.	Total Patients	Ears Total	Total Dry Ears By All Means			Total Furacin Failures
1	34	40	First course furacin	30		5
			Second course furacin	5		
			Other means		5	
2	11	15	First course furacin	6		6
			Second course furacin	3		
			Other means		2	
3	10	10		0		10
Grand total	55	65		44	7	21

TABLE 3. DATA ON 62 PERFORATIONS.

Group No.	Total No. Perforations	Excluded from Total No. Ears in Table 2	Total No. Perforations Failed to Close	Total No. Perforations Closed	Total No. Perforations Rupturing After Closure
1	38	1 (Failed to complete treatment)	1	37 (Includes one perf. closed twice)	5 (One of these perforations was closed a second time)
2	11 (Includes one ear with a double perf.)	5 (Two radical mastoid cavities. Three failed to complete treatment)	11	0	0
3	9	1 (Radical mastoid cavity)	9	0	0
4	4	0	0	4	0
	—	—	—	—	—
Grand total	62	7	21	41	5

Group 2 is made up primarily of those patients who obtained a dry ear without healing of the perforation. There were 11 patients in this group with 15 chronic running ears.

In this group six ears became dry with the initial course of furacin therapy, and three ears required a second course of treatment. In two additional ears there was a recurrence after furacin therapy. In one of these a dry ear was secured by the local use of a 5 per cent solution of glacial acetic acid in 95 per cent alcohol. The remaining one ear presented a rim of tympanic membrane, continuous purulent discharge, profound loss of hearing, and an intensely red, swollen and bleeding middle ear mucosa. A dry ear was secured by applying small amounts of trichloracetic acid to the middle ear mucosa overlying the promontory, as previously mentioned.

Group 3 is made up of those patients who failed to secure a dry ear with furacin or other solutions. This includes four ears of Group 2 and an additional 10 patients with unilateral involvement.

Group 4 consists of four patients with unilateral dry middle ears and perforated tympanic membranes who were not included in the grand total of 145 patients.

In considering the overall results with furacin therapy in Groups 1, 2 and 3 there was a total of 65 chronic discharging ears, of which 44 became dry with the initial or second course of furacin therapy for an incidence of 67.7 per cent. This total includes the two ears of Group 1 in which sensitivity developed but which eventually became dry.

In five ears of Group 1 and in two ears of Group 2 a dry ear was obtained by means other than furacin. Thus dry ears were secured in 51 out of a total of 65 chronic running ears for an incidence of 78.5 per cent successful results.

Sensitivity to furacin developed in three patients in Group 1 and in four patients in Group 3, there being no reactions in the patients of Group 2. The solution was not used in the patients of Group 4. Thus there was a total of seven sensitivity reactions out of a total of 55 patients for an incidence of 12.7 per cent. The only severe reaction occurred in a

patient who did not follow instructions to discontinue the medication when symptoms of sensitivity developed. With one exception the reactions occurred after the first week of treatment.

Since the patients in this series were seen at approximately weekly intervals, it was not possible in most instances to determine the exact number of days required to secure a dry ear with furacin in each patient. In Group 1 the two ears which developed sensitivity but which eventually became dry required 67 days and 127 days, respectively, to develop a permanently dry ear. During this time in both instances periods of moisture alternated with periods of dry ear until a permanently dry state was reached. If these two ears be excluded the remaining 33 ears required a total of 451 days to become dry. The average period of time required, therefore, was within 13.6 days. In like manner the nine ears of Group 2 in which furacin was successful required a total of 174 days to become dry. The average period of time for this group was within 19.3 days. The average period of time required to obtain a dry ear in both groups was within 14.9 days.

In analyzing the causes for furacin failures the following observations were made. In Groups 1, 2 and 3 there were 21 failures with furacin therapy. In seven of these patients with unilateral involvement a dry ear was secured by other conservative means. The furacin failures were due to an associated chronic suppurative sinusitis in two cases. It is likely that these ears would have failed to respond to any method of aural therapy without attention to the attendant sinusitis. In three ears there was a continuous discharge and central perforation. In five ears failure was due to sensitivity to furacin and in seven ears to recurrence of the otitis. In one patient with unilateral involvement, central perforation and continuous discharge a simple mastoidectomy had been previously performed elsewhere without benefit. In two ears a diagnosis of cholesteatoma was reasonably certain. In one ear of a child with central perforation and continuous dis-

charge the ear failed to become dry after furacin therapy, tonsillectomy and adenoidectomy, and submucous resection of the nasal septum.

b. Perforations. In Group 1 there were 34 patients with 38 perforations. In a patient with bilateral involvement one perforation closed, but the patient failed to complete treatment for closure of the remaining perforation. This one perforation has been excluded. In an additional patient with bilateral involvement one marginal perforation failed to close. Of the 37 perforations remaining, 36 were closed with the inital course of treatment. Subsequent disintegration of the scar occurred in five ears. In one of these ears the perforation was closed a second time. The remaining patients with four ruptured scars failed to return for a second attempt at closure. Thus 37 out of 38 perforations were actually closed with the initial or second course of treatment, and 34 out of 38 perforations remained closed when last observed.

In Group 2 there were 11 patients with 15 draining ears, two of which were radical mastoid cavities. These two ears have been excluded along with three patients with unilateral perforations who failed to complete treatment. In one ear there was a double perforation. This gives a total of 11 perforations none of which were closed.

In Group 3 there were 10 patients with nine perforations, one radical mastoid cavity being excluded. No perforations were closed in this group.

Group 4 consists of four patients with unilateral dry middle ears and perforated tympanic membranes which were not included in the grand total of 145 patients. All four perforations were closed in this group.

The combined perforations of Groups 1, 2, 3 and 4 total 62, of which 41 were closed with the initial or second course of treatment for an incidence of 66.1 per cent. There was subsequent disintegration of the scar in five ears, with one of these perforations being closed a second time; therefore, a total of 37 out of 62 perforations remained closed at the time of the last observation. This gives an incidence of 59.7 per cent

successful closure of all types of perforations under all conditions encountered in this study.

Table 4 lists the size and location of each perforation in Groups 1 and 4, the number of treatments required for closure of each, and the period of time the patient was observed after closure of the perforation (extended into 1952).

TABLE 4. GROUP 1.

Patient No.	Perforation	Number Treatments to Heal	Post-treatment Observations (Months)
1	Large inf. cent.	5	35
2	Large post. cent.	15	42
3	Large inf. cent.	15	35
4	Small inf. cent.	2	1
5	Mod. inf. cent.	7	39
6	Large ant. cent.	26	32
7	Mod. inf. cent.	4	1
8	Mod. post. cent.	7	1
9	R–Small post. cent.	4	6
	R–Small post. cent. (recurrence)	5	
	L–Large inf. cent.	20	
10	Mod. post. sup.	18	30
11	Large inf. cent.	25	22
12	Mod. post. cent.	9	26
13	Large inf. cent.	30	1
14	Mod. post. sup.	10	24
15	Mod. ant. cent.	5	1
16	Large ant. cent.	36	1
17	Large ant. and cent. attic	12	25
18	Mod. inf. cent.	14	22
19	Small ant. cent.	5	1
20	R–Large inf. cent.	29	13
	L–Mod. ant. cent.	28	
21	Mod. post. cent.	6	23
22	Mod. ant. cent.	12	13
23	Large post. sup.	10	20
24	Mod. post. cent.	10	2
25	Large ant. cent.	24	5
26	R–Mod. post. cent.	17	6
	L–Large post. sup. marg.	Failed	
27	Large post cent.	21	6
28	Mod. ant. cent.	15	1
29	R–Large inf. cent.	Excluded	
	L–Large inf. cent.	17	1
30	Mod. post. cent.	30	1
31	Mod. inf. cent.	7	5
32	Large post. cent.	26	1
33	Mod. inf. cent.	9	1
34	Large post. cent.	6	1
	Total	541	
	Average	14	

TABLE 4. GROUP 4.

Patient No.	Perforation	Number Treatments to Heal	Post-treatment Observations (Months)
1	Small post. cent.	2	1
2	Mod. inf. cent.	6	13
3	Mod. post. cent.	6	13
4	Small ant. cent.	3	4
	Total	17	
	Average	4.2	

Failure of the perforations to close was attributed to the marginal nature of the defect in four ears, large size of the defect in eight ears, and failure to obtain a dry ear in nine ears.

Disintegration of the scars was attributed to recurrence of the otitis media in two ears, and in the remaining three ears the cause was not determined unless due to vigorous blowing of the nose. In none of these cases was there a history of respiratory infection, trauma or swimming, and the ears remained dry.

It seems that the contraindications to closure of tympanic membrane perforations outlined by Dunlap and Schuknecht[131] are valid with one exception. These authors, as do others, advocate a dry ear before attempting closure of the perforation. I have not found this necessary in most cases of exacerbations of intermittent chronic otorrhea with nonmarginal perforations of the membrana tensa; in some cases of continuous otorrhea with the same character of perforations; and in one case of perforation of Shrapnell's membrane apparently representing localized attic disease without cholesteatoma formation. Adams[135] reported several cases in which he started closure of the tympanic membrane perforation at a time when the ear was actively discharging, and he was able to secure a healed perforation in all cases. The patients had intermittent otorrhea and perforation of the membrana tensa. In cases of continuous otorrhea one would not be justified in continuing efforts to close the perforation unless there was some evidence of diminishing discharge after a reasonable period of treatment.

In Group 1 closure of the perforation was started in 33 ears at a time when they were actively discharging, and of this number, closure was obtained in 32 ears for an incidence of 97 per cent. The one perforation which failed to close was of marginal character. In the remaining cases in this group a dry ear was obtained before closure of the perforation was undertaken.

INFLUENCE OF FURACIN ON CONTROL OF AURAL GRANULATIONS.

Since large granulations were removed by snare before furacin was started and acid applied to small visible granulations coincident with closure of the perforation, there was no opportunity to observe the influence of furacin on regression of this tissue. In four radical mastoid cavities (three patients) in which it was used in the postoperative care there was no apparent influence of furacin on controlling the granulations. Neither was there any deleterious influence on epithelization.

INCIDENCE OF ADENOIDS OR RESIDUAL ADENOID TISSUE AND DEVIATED NASAL SEPTA IN PATIENTS WITH CHRONIC OTORRHEA AND RESULTS OF TREATMENT OF THEIR AURAL CONDITION.

During the course of this study an attempt was made to evaluate carefully the degree of physiologic obstruction produced in the nasal cavity by deviation of the nasal septum. In like manner an attempt was made to evaluate the presence of adenoids or residual adenoid tissue following previous adenoidectomy. Major consideration was given to the mass of lymphoid tissue in the fossa of Rosenmüller and prominence of the lateral pharyngeal bands of lymphoid tissue. These observations have been tabulated in Table 5.

TABLE 5. DATA ON INCIDENCE OF ADENOID OR RESIDUAL ADENOID TISSUE AND DEVIATED NASAL SEPTA IN 55 PATIENTS WITH CHRONIG RUNNING EARS.

Group No.	Total No. Deviated Septa	Total No. Cases with Adenoids or Residual Adenoid Tissue	Total No. Cases with Adenoid or Residual Adenoid Tissue Presenting Deviated Septa	Total No. Cases with Deviated Septa Without Adenoids or Residual Adenoid Tissue
1	13	24	10	3
2	6	9	5	1
3	4	7	3	1
Grand total	23	40	18	5

It was considered that there were 13 obstructing deviations of the nasal septum in Group 1, six in Group 2, and four in Group 3. This gives a total of 23 deviated septa seen in 55 patients for an incidence of 41.8 per cent.

In Group 1 there were 24 patients with adenoids (14 cases) or residual adenoid tissue (10 cases). In this total number of 24 cases the nasal septum was deviated in 10. The septum was deviated in three patients without a significant amount of adenoid or residual adenoid tissue. In Group 2 there were nine cases with adenoid (five cases) or residual adenoid tissue (four cases). In this total number of nine cases the nasal septum was deviated in five patients. In one case the septum was deviated without a significant amount of adenoid or residual adenoid tissue. In Group 3 there were seven patients with adenoids (four cases) or residual adenoid tissue (three cases). In this total number of seven cases the septum was deviated in three cases. In one case the septum was deviated without a significant amount of adenoid or residual adenoid tissue.

In summarizing these observations it may be seen that there was a total of 40 patients with adenoids (23 cases) or residual adenoid tissue (17 cases). In 18 of these 40 cases there was an associated deviation of the nasal septum. In five patients a deviated septum was present without adenoid or residual adenoid tissue.

It is a matter of interest to consider the clinical results obtained in these patients. In Group 1 there were 27 patients with 32 running ears who had a significant amount of adenoid or residual adenoid tissue, deviated septum, or both. This total includes one ear in which there was recurrence of the otitis with a dry ear being obtained a second time. A dry ear was secured in all of these 32 ears by furacin or other means for an incidence of 100 per cent. There were 31 perforations in this total of 32 ears, including one twice closed and excluding one perforation in the patient who failed to complete treatment. Of the 31 perforations, 30 were closed, the one perforation failing to close having a marginal character. Sub-

sequent rupture of the scar occurred in five ears. In one ear the perforation was closed again, but the remaining four patients failed to return for a second attempt at closure. It is difficult to say what part if any the lymphoid tissue and deviated septa played in the disintegration of the scars in these ears.

In Group 2 there were 10 patients with 14 running ears who had adenoid or residual adenoid tissue, deviated nasal septa, or both. A dry ear was secured by furacin or other drugs in 10 ears for an incidence of 71.4 per cent. No perforations were closed in this group.

The combined results of Groups 1 and 2 show that there was a total of 46 draining ears with adenoid or residual adenoid tissue, deviated nasal septa, or both. In this total of 46 ears, 42 became dry for an overall incidence of 91 per cent. It is impossible to say what part the adenoid or residual adenoid tissue and deviated septa played in the failures of Group 3 in those cases in which they were present.

ROLE OF TONSILLECTOMY AND ADENOIDECTOMY IN THE TREATMENT OF CHRONIC SUPPURATIVE OTITIS MEDIA.

Another point developed in this study was the role of tonsillectomy and adenoidectomy in the treatment of chronic suppurative otitis media. As a part of the treatment for chronic otorrhea the tonsils and adenoids had been removed in 16 patients in Group 1, in five patients in Group 2, and in three patients in Group 3. Thus 24 out of 55 patients, or 43.6 per cent, had been subjected to tonsil and adenoid surgery. In three patients the ears became dry after operation, but the otorrhea eventually recurred. In 21 patients, or 87.5 per cent, removal of the tonsils and adenoids had no influence whatsoever on the course of the otorrhea. In 19 of these patients surgery had been performed before the age of 10 years.

These results do not necessarily condemn tonsillectomy and adenoidectomy in the treatment of chronic otorrhea. At least three vital points must be taken into consideration. The age of the patient at the time of surgery must be considered. Fow-

ler[142] stated that unless tonsils and adenoids are removed soon after chronicity develops or recurrence threatened, and before the child's ninth year, the results are doomed to failure in most instances. My own previous experience is in essential agreement with this conclusion. The majority of operations in this series were performed before the tenth year, yet most of them failed to secure a dry ear. This leads to a second important factor. The method of removing tonsils and adenoids is of paramount significance. The most complete adenoidectomy possible consists in the use of adenotome, curettes, or both, followed by removal of the adenoid tissue in each fossa of Rosenmüller, the lateral pharyngeal bands of lymphoid tissue, and the nodules of lymphoid tissue on the posterior pharyngeal wall. This is best accomplished by use of punch forceps after elevation of the soft palate. The technique is essentially that recently described by Reeves and Brill.[143] It did not appear from the examination of these patients that this method had been routinely followed in their surgery. A third most important factor, and one that is perhaps too infrequently considered, is the inherent nature of the pathologic process responsible for the chronic otorrhea. In cases of osteitis, mastoiditis and cholesteatoma, any adenoidectomy, however well performed, regardless of age, is likely to fail as a therapeutic procedure for the chronic running ear.

HEARING BEFORE AND AFTER CLOSURE OF THE TYMPANIC MEMBRANE PERFORATION.

Audiograms were made before and after closure of the tympanic membrane perforation on 22 patients with 24 draining ears in Group 1. The results were computed by averaging the decibel changes in the frequencies 512 through 2,048, inclusive, in the first pre- and post-treatment audiograms. In six ears in which there was both gain and loss of hearing acuity the average for each was computed and the difference recorded as the average decibel gain or loss according to which prevailed. The results have been recorded in Table 6.

TABLE 6. AVERAGE DECIBEL CHANGE IN 24 EARS BEFORE AND AFTER CLOSURE OF THE TYMPANIC MEMBRANE PERFORATIONS (GROUP 1).

Patient	Aver. Db. Gain		Perforation		Aver. Db. Loss
1	17.5		Large inf. cent.		
2	26.2		Large post. cent.		
3	21.2		Large inf. cent.		
4	10.0		Large ant. cent.		
5			Mod. inf. cent.		2.5
6			Small post. cent.	-R-	20.0
			Large inf. cent.	-L-	23.7
7	20.0		Large inf. cent.		
8	46.2		Mod. post. cent.		
9	17.5		Mod. post sup.		
10	15.0		Large ant. cent.		
11	10.0	-R-	Large inf. cent.	-R-	10.0
	15.0	-L-	Mod. ant. cent.		
12	7.5		Mod. ant. cent.		
13	11.2		Large post. sup.		
14	27.5		Mod. post. cent.		
15	22.5		Large ant. cent.		
16			Mod. post. cent.	-R-	5.0
			Left failed to close		
17	2.5		Large post. cent.		
18	10.0		Mod. ant. cent.		
19	1.6		Mod. post. cent.		
20			Mod. inf. cent.		7.5
21	11.2		Large post. cent.		
22	20.0		Large post. cent.		

In the total of 24 ears, 15, or 62.5 per cent, showed a gain in hearing acuity of 10 or more decibels. In three ears, or 12 per cent, the gain in hearing acuity was less than 10 db. In five ears or 20.8 per cent, there was a further impairment of hearing. Two of these ears showing a loss were in a patient with bilateral involvement. In the remaining three ears the hearing loss was less than 10 db. In one ear, or 4.1 per cent, the average decibel gain in hearing acuity equalled the average decibel loss (Patient 11, Table 6).

A consideration of the overall results shows that improvement in hearing acuity of significant degree occurred in the majority of patients. Dunlap and Schuknecht[131] stated that if the ossicular chain be intact, hearing is almost certain to be improved. In their experience better auditory acuity resulted from closure of defects beneath the umbo. In the 15 ears in which hearing acuity was improved by 10 or more decibels

the perforations, ranging from moderate to large in size, were classified as anterior and posterior central in five ears each, inferior central in three ears, and posterior superior in two ears. The greatest average gain in hearing acuity was seen in the patients with posterior central perforation. The average gain in this group, however, was raised considerably by the gain of 46.2 db in one ear. If this ear be excepted, then the average gain in hearing acuity for all types of perforations was essentially the same.

Fowler[142] studied the results of repeated audiometric tests made over a period of years on a large group of clinic children treated for chronic suppurative otitis media. He concluded that while treatment produced improved hearing in individual cases, the average results were disappointing. In the majority of cases a dry ear and healed perforation should afford the maximum gain in hearing acuity. It seems logical, therefore, that all efforts should be bent to secure a healed perforation whenever this possibility seems likely.

CASE REPORTS.

In the following representative case reports taken from Group 1 only pertinent facts pertaining to each case have been recorded. A chronic external otitis was frequently associated but was usually of mild degree. Closure therapy refers to application of trichloracetic acid to the edges of the perforation, granulations or base of the granulations when these were removed. At the time of the last observation a dry ear and healed perforation were still present unless notes are made to the contrary.

Case 1: R. L., a white female, aged 41 years, referred by Dr. K. P. Turrentine, first observed March 11, 1948. Right ear. Duration since September, 1947, when the patient sustained a traumatic injury to the right ear superimposed on a chronic external otitis. Drainage intermittent from September to December, 1947; constant, foul and moderate in amount since December; purulent. Chronic bronchial asthma. *Examination:* Large posterior central perforation with small granulations in the middle ear. Staphylococci cultured. Hypertrophic rhinitis. Nasal septum deviated to the right. Few eosinophiles in nasal smear. *Course:* Furacin and closure therapy started March 11, 1948. Ear dry within 14 days, granulations healed within three weeks, and perforation closed after 15 treatments. Last observed May 2, 1952.

Case 2: J. B., a white female, aged 18 years, first observed July 14, 1948. Left ear. Duration since age of three months, following whooping cough and pneumonia. Drainage intermittent, profuse and occasionally foul and blood streaked. T and A in childhood and radium therapy to nasopharynx one year before without benefit. Chronic hay fever. *Examination:* Large inferior central perforation with lower two-thirds of the malleus handle devoid of covering but without gross necrosis. Few eosinophiles in nasal smear. *Course:* Failed to return until Sept. 1, 1948, when furacin and closure therapy were started. Ear dry within seven days. Perforation closed after 15 treatments. Last observed Feb. 23, 1952.

Case 3: F. O., a white female, aged 28 years, referred by Dr. F. Fuller, first observed Oct. 25, 1948. Left ear. Duration since childhood. Drainage intermittent, profuse, foul at all times; mucopurulent. *Examination:* Large anterior central perforation. Tonsils and adenoids small. *Course:* Furacin started Nov. 2, 1948. Ear dry within seven days. Closure therapy started Nov. 16, 1948. Perforation healed after 26 treatments. Last observed April 12, 1952.

Case 4: G. M., a white male, aged 18 years, first observed Feb. 2, 1949. Bilateral. Right ear draining one year. Left ear draining since infancy. Drainage from right ear intermittent until four weeks ago. Since then it had been continuous, foul, scant; purulent. Drainage from left ear intermittent until one year ago. Since then it had been continuous, foul, profuse; purulent. Simple mastoidectomy left side in 1941 and T and A in 1946, both without benefit. *Examination:* Small posterior central perforation right. Large inferior central perforation left. Small granulations both middle ears. Nasal septum deviated to the left. *Course:* Closure therapy started Feb. 8, 1949. Furacin started Feb. 19, 1949. Right ear dry within seven days and left ear within 30 days. Perforation right healed after four treatments. Perforation left healed after 20 treatments. Discharged Aug. 31, 1949. Returned Jan. 13, 1951, with acute exacerbation of a chronic external otitis right and draining right middle ear. Same character of perforation and discharge as noted Feb. 2, 1949. No change in left ear. *Course:* Furacin started June 13, 1951. Ear dry within seven days. Closure therapy started June 20, 1951. Perforation healed after five treatments. Last observed Dec. 28, 1951.

Case 5: L. E. C., a white male, aged 10 years, referred by Dr. W. E. Keiter, first observed June 6, 1949. Left ear. Duration since age nine months. Attributed to recurrent acute abscesses in the ear. Drainage intermittent, moderate in amount, foul four or five years; mucopurulent. T and A three years before without benefit. *Examination:* Large inferior central perforation with lower half of malleus handle devoid of covering but grossly without necrosis. Residual adenoid tissue. *Course:* Furacin and closure therapy started June 8, 1949. Each dry within 28 days. Recurrence of moisture July 22, 1949. Periods of moist ear alternated with periods of dry ear until the perforation was closed on Dec. 23, 1949, after 25 treatments. Last observed Nov. 20, 1951.

Case 6: A. D., a white male, aged seven years, first observed Aug. 5, 1949. Right ear. Duration one year. Attributed to recurrent abscesses in the ear with repeated myringotomies. Drainage intermittent until two months before when it became continuous, scant, foul; purulent; severe bleeding from the ear three days before, necessitating packing by the family physician. *Examination:* Large mass of granulations filling external auditory canal. Tonsils and adenoids enlarged. Failed to return until Sept. 7, 1949, when granulations were removed by snare. Moderate size

anterior central perforation. *Course:* Furacin and closure therapy started Sept. 7, 1949. Ear dry within 14 days. Perforation closed after five treatments. Last observed Oct. 26, 1949.

Case 7: N. C., a white male, aged 13 years, first observed Aug. 12, 1949. Left ear. Duration since age seven months. Attributed to recurrent acute abscesses in the ear with repeated myringotomies. Drainage continuous, varied in amount, foul at times; purulent. T and A previously without benefit. *Examination:* Large anterior central perforation. Nasal septum deviated to the right. Residual tonsil and adenoid tissue. Bilateral chronic suppurative maxillary and ethmoidal sinusitis. *Course:* Furacin started Aug. 15, 1949. Ineffective after two weeks. Proetz displacement, bilateral antral lavage, and closure therapy started Aug. 27, 1949. Discharge became mucopurulent, odorless, scant. Visits irregular. Periods of moist ear alternated with periods of dry ear until the ear was permanently dry, May 5, 1951. Perforation closed after 36 treatments at irregular intervals. Last observed July 3, 1951.

Case 8: Z. J. C., a white female, aged six years, referred by Dr. Rachel Davis, first observed Aug. 31, 1949. Right ear. Duration since infancy. Attributed to repeated acute abscesses in the ear. Drainage intermittent, varied in amount; mucopurulent. *Examination:* Moderate size inferior central perforation. Tonsils and adenoids enlarged. *Course:* Furacin and closure therapy started Sept. 2, 1949. Ear dry within seven days. Perforation healed after 14 treatments. T and A May 26, 1950. Last observed Nov. 5, 1951.

Case 9: C. T. J., a white female, aged 43 years, referred by Dr. S. Parker, first observed June 25, 1950. Left ear. Duration 17 years. Attributed to repeated acute abscesses in the ear. Drainage intermittent until six months previously. Since then it had been constant, moderate in amount; purulent. T and A six months after onset and simple mastoidectomy 12 months later, both without benefit. Food allergy. *Examination:* Moderate size anterior central perforation. *Course:* Furacin and closure therapy started June 29, 1950. Ear dry within 60 days. Perforation closed after 12 treatments. Last observed Oct. 31, 1951.

Case 10: F. M. T., a white male, aged 12 years, referred by Dr. J. Bower first observed Nov. 8, 1951. Right ear. Duration three months following swimming. Drainage intermittent, moderate in amount; mucopurulent. *Examination:* Moderate size inferior central perforation. Ear dry. Nasal septum deviated to the left. Tonsils and adenoids enlarged. *Course:* Closure therapy started Nov. 13, 1951. Recurrence of otorrhea Dec. 7, 1951, following acute external otitis. Furacin started Dec. 7, 1951. Ear dry within seven days. Perforation closed after seven treatments. Last observed May 28, 1952.

Case 11: M. H., a white female, aged 34 years, referred by Dr. J. Morris, first observed Oct. 23, 1951. Left ear. Duration 18 months following a severe cold with an acute middle ear abscess. Drainage intermittent at first. During the preceding eight months the drainage became continuous, profuse, foul, blood streaked; frank bleeding one week before; purulent. *Examination:* Large granulations removed by snare Oct. 25, 1951. Large posterior central perforation. Gram-negative bacilli and Gram-positive cocci cultured. Cholesterol crystals present. *Course:* Furacin and closure therapy started Oct. 25, 1951. Ear dry within 14 days. Granulations healed Dec. 8, 1951. Perforation healed after six treatments at two-week intervals. Last observed Feb. 22, 1952.

SUMMARY.

A total of 145 patients with chronic running ears (incidence 3 per cent) were seen in office practice over a three-year period. These patients were studied to such an extent as cooperation of the patient, economic factors and local facilities would permit, and the results have been recorded herein. Salient points regarding the etiology of chronic suppurative otitis media, cytology of aural secretions, significance of aural granulations and cholesterol crystals, and healing and method of closure of the perforated tympanic membrane have been presented from the literature and personal experience. The bibliography concerning the topical application of certain digestant, chemotherapeutic and antibiotic agents in the treatment of chronic otorrhea has been included.

Out of the total of 145 patients, 55 cooperated in the use of anhydrous furacin ear solution. The results of treatment have been discussed in terms of success or failure to obtain a dry ear, healed perforation and improved hearing acuity.

DISCUSSION.

It is apparent from this study that the majority of dry ears were secured in those patients with intermittent discharge of mucopurulent character and moderate to large size perforations of the membrana tensa. It has long been recognized that this class of patients respond best to conservative treatment of almost any nature. The significance of these results, however, lies in the fact that 49 out of 55 patients (89 per cent) had been previously treated by one or more otologists with failure to obtain a permanently dry ear.

The results obtained with furacin therapy in this series of patients compare favorably with the results reported in the literature from the use of other digestant, chemotherapeutic and antibiotic agents. I have previously used sulfanilamide and sulfathiazole powder alone or in combination with zinc peroxide powder, "dalyde," glycerite of hydrogen peroxide and penicillin solution in the local treatment of chronic running ears. The results obtained with furacin therapy have been superior to those obtained with the above agents in spite of the high incidence of local reactions.

Perforations which were surrounded by a good margin of tympanic membrane healed most readily. One perforation of Shrapnell's membrane healed. The healing response manifested by the tympanic membrane was slow, rapid or failed altogether, there being considerable variation in individual response. Denudation of the handle of the malleus without gross necrosis did not preclude healing of the perforation, but the response tended to be slow. No perforation of marginal character was closed.

When the tympanic membrane perforation healed, the resulting cicatrix so closely resembled the surrounding drumhead that it was impossible to outline the original perforation. After several months, however, such delineation was possible because of the translucency which developed in the scar. In several instances when the perforation was healed to an aperture of a millimeter or so, a thin, extremely delicate scar developed which closed the remaining defect in whole or in part. It was obvious that this delicate membrane was not durable, and it was therefore repeatedly ruptured in order to produce a more satisfactory cicatrix for the remaining perforation. In all instances this was accomplished, but the time required was much longer than one would expect for the closure of such a small defect. A number of patients have been followed for several years, and in most of these chalk deposits have been noted in the scars. In some instances no portion of the scar tissue could be visualized because of the extensive nature of the chalk deposits.

CONCLUSIONS.

Anhydrous furacin ear solution is a valuable adjunct in the treatment of the nondangerous form of chronic suppurative otitis media. The incidence of sensitivity reactions is higher than that previously reported in the literature with its use in the treatment of aural infections. The continuous method of topical application of a chemotherapeutic agent over a long period of time is highly satisfactory. Dry ears may be obtained in many cases when local cleanliness is a minor part

The anhydrous furacin ear solution used in this study was supplied in part by Eaton Laboratories, Norwich, N. Y.

of the treatment. Cholesterol crystals in the aural discharge of chronic running ears is a poor indication of the presence or absence of cholesteatoma of the middle ear. Dry ears and healed perforations may be secured in many patients in which there is associated nasopharyngeal lymphoid tissue, deviated nasal septum, or both. Removal of tonsils and adenoids for the treatment of chronic suppurative otitis media should be done only after consideration of the inherent nature of the pathologic process responsible for the otorrhea, age of the patient, and method of performing adenoidectomy.

Trichloracetic acid in 50 per cent solution is an excellent cauterizing agent for stimulating tympanic membrane repair. Actively discharging ears of the nondangerous type do not preclude its use providing there is evidence of cessation of the discharge after a reasonable period of time. Healing of a perforation is no guarantee against subsequent rupture of the scar. Closure of tympanic membrane perforations is attended by improved hearing acuity of significant degree in the majority of cases.

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Kinston Clinic.

HEARING IMPAIRMENT IN PRESCHOOL-AGE CHILDREN.*

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Much attention is at present being given to the status and needs of the preschool-age child with hearing impairment severe enough to delay or retard normal behavior and development, particularly the development of language and speech. For obvious reasons, a child learns to talk because and as he hears; and if hearing is impaired enough to interfere with the normal development of language and speech, a child will need special help. The nature and extent of this help depends upon: *1.* the cause, nature and extent of the hearing impairment; *2.* his physical, intellectual and social maturity; *3.* the status of his communicative skills. These are the facts to be ascertained in diagnostic procedures. When they are known, a program of training can be outlined according to the needs of the particular child.

A recent study of 572 infants and preschool-age children has produced some interesting data with far-reaching implications. This group of children represents the array that was seen for otologic and audiologic study in an 18-month period at a medical diagnostic center. During this period, some of these children were retested, others have been retested since and the retest findings are entirely commensurate and consistent with the original findings. These children were seen clinically because they had or were suspected of having serious communicative problems. The group represents a wide geographic distribution.

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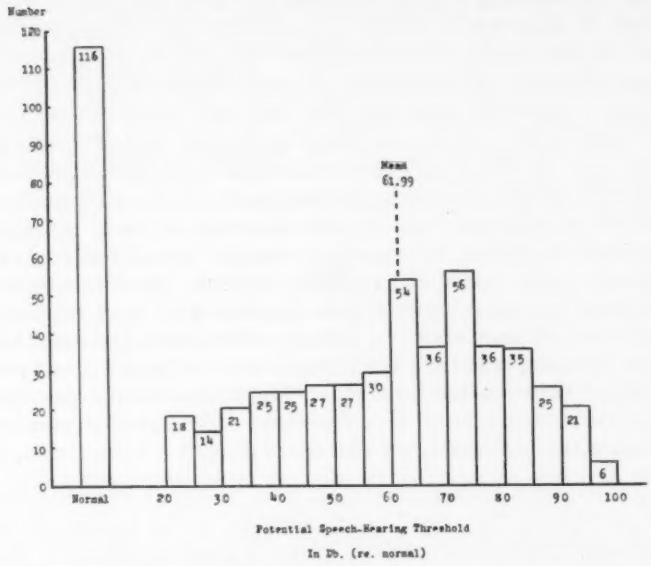

Fig. 1. Preschool-age children (572 cases) with serious communicative problems. Age range: four months to five years. Mean age: two and nine-tenths years.

Information like that presented in Figure 1 was tabulated at three different stages, when there were 167, 378, and finally 572 cases. It would seem significant that the mean level of impairment has not varied by as much as 1 db with tripling the number of cases, nor has there been any significant variation in the percentage of children in like audiologic categories according to the level of impairment (see Fig. 2).

These children ranged in age from four months to five years. The youngest were seen largely for one of two reasons: audiologic appraisal was requested because the children were up for adoption; or a child had had a serious illness, a meningitis or encephalitis, which offered a predilection for impaired hearing. The apparent etiologies were many and varied and run the gamut of prenatal, neonatal and postnatal problems. Many of the cases were referred by pediatricians

and had received a pediatric work-up. All were given a thorough otologic examination, and medical case histories were as complete as they could be made. Many were given careful developmental examinations; all were observed carefully in terms of developmental behavior. All were given as complete an audiologic work-up as fairly extensive clinical facilities can provide. When indicated, neurologic and psychiatric consultations were provided. In audiologic terms, all were observed in play activity; careful observations were made of general responses to measured sounds; speech-hearing responses were measured whenever possible. Routine clinical pure-tone audiometry was not feasible with most of these children; all were tested by galvanic skin-resistance audiometry. Masking was used whenever it was indicated by an imbalance between the two ears; bone-conduction audiometry was done occasionally (*i.e.*, for cases of bilateral atresia, or other structural developmental anomalies).

Information plotted on the abscissa in Fig. 1 was obtained by averaging the pure-tone thresholds in the better ear at 500, 1,000 and 2,000 cycles. There is ample evidence in the literature of the field to suggest a good warrant for this procedure, relative to the particular interpretations made in this study. Whether the measurements by PGSR audiometry represent specific threshold acuity is problematical; the procedure and the findings have been validated in various ways, including test-retest of a significant number of children of comparable age and status and with normal-hearing children. Where error exists, it lies in not reaching the child's thresholds for pure tone. It is safe to say that the hearing of these children is no worse than is represented in these findings; in some, without doubt, the measurement did not reach threshold acuity.

One-fifth of the group (116 children) was found to have normal auditory function; that is, the function of the auditory end-organ was found to be normal, and any difficulty in communication could not be attributed to an impaired hearing mechanism. In this group were postmeningitic, motor-palsied, cerebral degenerate, slow-learning, dysphasic, schizoidal and

psychologically disturbed children; some belong in the category of *delayed speech, i.e.,* for various reasons they had not developed speech but probably would. It is worth noting that many of these children acted as though they did not hear; yet the problem centered elsewhere than in the auditory mechanism, and was attacked accordingly. This group of children is not considered further in the present discussion.

Four-fifths of the group (456 children) had impaired hearing. Of these, 18 had conductive lesions of moderate degree; the others had nerve-type or mixed-type lesions. The causal relations, as well as they can presently be determined or estimated, range from biologic maldevelopment and prenatal infection through a wide variety of postnasal diseases and traumas. Approximately one-third of the problems are ascribable to prenatal problems or birth trauma; roughly, two-thirds to neonatal or postnatal difficulties. In the prenatal group there were found factors of heredity, virus infection in the mother, toxemia in pregnancy and uterine disturbances. Hereditary factors that may be readily demonstrated (*i.e.,* two children in the same generation, or one child in each of two ascending generations, both involved from birth) are infrequent in occurrence (14 children). The postnatal problems include a presumption of the effects of cyanosis, the Rh factor, and other evidences of neonatal trauma; by far the largest group involved virus-type infections, particularly in the first year of life. Of the total group of affected children, at least 25 per cent must be labelled *undeterminable* for cause; in this group, one-third presented totally negative histories, while two-thirds presented so much data that it was impossible to make even a reasonable guess as to specific cause.

SPEECH-HEARING THRESHOLD POTENTIALS.

Of particular significance is the estimated speech-hearing threshold potentials of these children. A speech-hearing threshold is the level of intensity at which speech sounds are just intelligible to the listener. This function involves listening, language recognition, and intelligence and represents the level of threshold acuity at which the residual hearing is used. For the very young child, this must be a judgment in terms of

the clinical findings and psychoacoustic facts. The clear perception of speech sounds—and, therefore, a major pathway for learning language — involves reasonably well-balanced hearing across a wide acoustic range. A child may hear within a part of the range of conversational sounds, but not all; he may hear little of ordinary speech with a damaged hearing mechanism, but a great deal with amplification. He hears quite differently with a conductive, nerve-type or mixed-type impairment. How he hears largely governs the interpretative facility with which he may learn to understand. For these reasons, it is believed that determination of the unaided speech-hearing threshold potential, with respect to the type of hearing impairment involved, is the most important single factor in the prognosis and handling of these children.

The 456 children with impaired hearing were grouped into four categories according to the level of impairment, as delineated in Fig. 2:

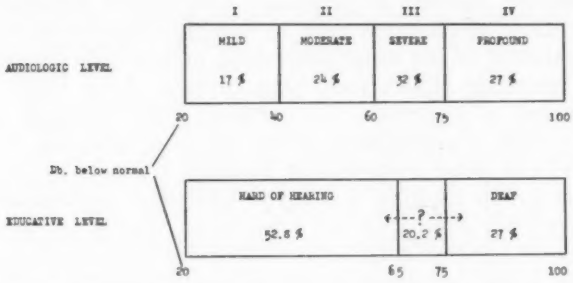

Fig. 2. A comparison in audiologic and educative terms, relative to levels of hearing of 456 affected children.

Group I (17 per cent)—These are the mild-loss cases with unaided threshold potentials of 20 to 40 db below normal. This level of loss means roughly that an average conversational voice can be intelligible at a three-foot distance.

Group II (24 per cent)—These are the moderate-loss cases with unaided speech-hearing threshold potentials of 40 to 60 db below normal. For them, a loud conversational voice can be detected at a three-foot distance.

Group III (32 per cent)—These are severe losses, with unaided threshold potentials between 60 and 75 db below normal. This level means roughly that a loud conversational voice does not come through; only loud environmental noises will arrest attention.

Group IV (27 percent)—These are profound losses, with unaided speech-hearing potentials between 75 and 100 db below normal. At this level only the very intense, usually vibratory noises of the environment can be detected.

INTERPRETATION.

It is important to note that the mean threshold potential for the entire group of 456 affected children was only 61.9 db below normal; even more significant is the fact that no child had a total hearing loss. It is commonly stated, particularly in regard to children in our schools for the deaf, that there is much total deafness. Present clinical evidence indicates that in this age of chemotherapy there are relatively few children who are totally deaf, so far as complete dysfunction of the auditory mechanism is concerned. On the other hand, there are without doubt a great many school-age children who are functionally deaf, because they have not had the opportunity to use their residual hearing. Hearing is a function of the auditory end-organ; listening, or language decoding, or the perceptive use of hearing in communication, is a function of the cortex and is a learned process.

Children with speech-hearing threshold potentials down to about 75 db below normal can make use of audition as the major pathway for language comprehension, with visual stimuli as adjuncts. They can be helped by a good modern wearable hearing aid. It has been repeatedly demonstrated that with the early use of hearing aids, the children in Groups I and II can learn to interpret language through audition alone and can function at a dull normal level of intelligibility. With good handling, the children of Group III can learn to function with amplification at a serviceable level, *i.e.*, the equivalent of a mild hearing loss. It takes about two years of constant exposure to normal language and speech for the young child

to develop useful auditory patterns. As with most two- and three-year-olds, this development is best centered around the home and the daily activities of living. By the time they are four or five years of age most of the children in Groups II and III will be ready for intensive training in language skills, that is, in developing patterns of recognition and correlation (time order, structure, syntax) in terms of verbal symbols and training will vary markedly for each child. The educative goal for the children in Groups I, II and III is a regular school with whatever appropriate special help in communicative skills and language is needed. For many in Group III this may be achieved only after a period of language instruction in a special class, or school; for some who are slow to learn or for whom the home environment is not geared to help them, it may not be achieved at all. These three groups compose 73 per cent of the total in this study and, with the obvious exceptions noted in Group III, they can be labelled *hard-of-hearing* children for educative purposes.

On the other hand, the children in Group IV with profound losses, more than 75 db below normal, must learn to communicate principally by use of vision, with audition as a limited adjunct. It has been demonstrated, however, that most children in this group will comprehend language best with a combination of visual and auditory cues. With only a remnant of potential hearing, most children can make some profitable use of amplification *if the experience is begun early enough.* While such use of amplification can rarely bring into being independent auditory patterns, it can contribute significantly to vowel comprehension, awareness of voice and intonation, and to the child's awareness of his environment of sound. The children in Group IV will usually require intensive, long-term special education in a school for the deaf.

NEEDS OF THE VERY YOUNG CHILD WITH IMPAIRED HEARING.

Important and fundamental as are the psychoacoustic measurements, much more is involved in appraising and meeting the needs of the child with a handicapping hearing impairment. Quite as important are the data on and the evaluation of age at onset, etiology, physical development, social matur-

ity, home environment, parental attitudes, insight and handling; the effect of indicated medical and surgical procedures. One is never concerned with ears, but with a whole, behaving, developing child. The most important thing about hearing impairment is that it seriously interferes with the child's ability to communicate normally, and, therefore, interferes with a variety of conscious and unconscious adjustments that are essential to learning and to reasonably normal behavior. It is this communicative barrier that needs to be surmounted as rapidly as possible.

The factors necessary in working with the young child with impaired hearing include auditory training (experience) with or without a hearing aid, speech (lip) reading, speech training, careful presentation of language principles to be mastered, and careful guidance in behavioral, developmental terms. Auditory training has been employed with intent for a century and a half; speech reading has been known and taught for almost as long; speech training is at least as old as ancient Greek civilization. There have been schools for the deaf in this country since the beginning of the nineteenth century, and some sort of educative programs for the hard-of-hearing child for the past 25 to 30 years. The only really new tools are the modern hearing aid—whether it be a master aid of wide acoustic scope, a group aid or an individual wearing aid—and the development of diagnostic techniques that enable one to measure with some care the auditory function of very young children.

These new tools, however, have changed the whole complexion of the problem. Prior to the development of adequate hearing aids (it is assumed that all children suspected of impaired hearing should receive the benefits of careful medical diagnosis and all indicated medical and surgical treatment before any question of long-term handling is raised), a child with a 50 db hearing loss was as much out of touch with the hearing world as was a child with a 90 db hearing loss, and both were *deaf* with regard to auditory function in language. In most instances they were educated in the same way and in the same school. Since it was impossible to test accurately

the hearing of the infant or young child, no definitive tests were attempted until he entered school (five to six years), and his feet were firmly set on the path of deafness. Even at school age, any measurement of auditory function was of dubious accuracy, for techniques were largely dependent upon the perceptive responses of a mind that had never perceived sound and for whom the neurologic and psychologic functions and structures were set in nonauditory terms. Modern electronics has made possible the early appraisal of the level of hearing impairment in the infant and young preschool-age child and the initiation of an appropriate program, planned to stimulate language and speech development in the child's most formative years with the use of audition, the most important pathway to the development of the language functions of the mind.

SOME BASIC CONCEPTS.

Ten basic audiologic concepts have been established within the past few years. None of these is new in itself, but each plays a significant part of a unified whole, the aggregate of which can materially change the child's future.

1. Hearing rehabilitation is a many-sided cooperative endeavor involving the pediatrician, the otologist, the clinical audiologist, the psychologist, the teacher and, above all, the parent. It cannot be fully effective except as this group learns to work as a team.

2. Communication in these children is an entity, involving acoustic, linguistic, visual, behavioral, developmental sensory-motor and social elements which are contributory aspects of the whole child. Hearing, speech and language cannot be isolated from one another or divorced from the overall developmental processes.

3. Treatment and training should be based on a full diagnostic appraisal which includes an early measurement of the amount of residual hearing and, when possible, the child's ability to use it.

4. Treatment and training should be started as early as possible in the child's life. The period from 18 to 30 months

seems best. The child between the ages of two and five years is at his peak as a language-learning individual; never again will he exhibit such a state of readiness, need and desire for the acquisition of language and speech. So far as the tools of communication are concerned, the child's career does not begin at school, but in infancy.

5. With appropriate handling, even children with a profound impairment can learn to talk and participate in normal communication.

6. The majority of children with impaired hearing have a great deal of residual hearing and can utilize amplified sound to the utmost, providing this use is started at an early age and the effect made an integral part of the developing mind. Even the child with a profound loss can benefit to some degree from amplification.

7. Wearable hearing aids make sound louder and provide the means for putting the child in contact with sound through all his waking hours. Children seem to make the best adjustment to a wearable aid between two and three years of age. Each child must be timed according to his readiness and needs as a developing person. A serious error is often made in waiting too long, until the child is five or six years old, when his best period for language learning is past.

8. With any particular child who has a handicapping hearing impairment, the question is not, "Is special training necessary?" but "How much and what kind of special training is necessary?" Some special handling is always necessary at home and at school. Sometimes special work in connection with a regular nursery school or elementary school is indicated; sometimes a special day or residential institution seems best. This is a task for careful audiologic-educative determination.

9. Most children develop best in a situation that is the closest approach to normalcy yet which offers means for meeting the special needs of the child.

10. Parent understanding and parental guidance are the keys to early steps in working with the child with a severe hearing impairment. Progress is usually made in direct proportion to their understanding and acceptance of the problem with which they are faced. They need to understand how communication develops, and how they can and must stimulate it in the minute-by-minute experiences of the child. They must be shown how to communicate clearly and simply at short distance, using the same vocabulary over and over in a wide variety of situations, until meaningful relations are grasped and the child begins to relate and store them, and eventually to reproduce them in his own speech. They must learn how to anticipate communicative requirements and to expand the child's vocabulary after initial steps have been taken. There is always parental uneasiness in facing the daily problems related to the child, and parents need support and reassurance.

Within this framework, there are two streams of thought which have special implications for medicine and education: *1.* although each child is a unique individual, with his own special needs in communicative development, generalized methods can be made to work as the professional team learns to work together; *2.* the utilization of modern electronics makes possible an emphasis on hearing, not deafness, in the prospectus of the child's future. There is now a large group of veteran hearing-aid users under the age of six, giving self evidence that the child with a serious hearing impairment can and does learn language and speech as does the normal-hearing child, if an appropriate program is launched early enough and is followed through in the formative years.

"GREEN-BOTTLE FLY WORM" INFESTATION OF THE EAR. MYIASIS OF THE EAR CAUSED BY PHAENICIA SERICATA

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Myiasis is the term used to signify the diseases or symptoms produced by fly larvae when they feed and live parasitically in the tissues of man or other mammals. The infestation of sheep and cattle creates quite a serious problem in some areas, particularly in the warmer climates. Great losses of live stock may result.

Of the many species of flies causing myiasis probably the most common in the United States are the green-bottle fly worm *(Phaenicia sericata)* and the screw worm *(Cochliomyia macellaria)*.

Dr. Maurice T. James[1] in a monograph on "The Flies That Cause Myiasis in Man" (1947) speaking of the green-bottle fly worm states that the female fly deposits her eggs in a mass on or near food substance. The larva undergoes three molts; after a feeding period of one and one-half to nine and one-half days, it seeks a favorable place in the soil to pupate . . . The common breeding medium is carrion, although larvae have been reared from manure and from garbage. This species is frequently attracted to ill-smelling sores, and to soiled wool, and is one of the principal sheep-maggot flies in the British Isles, South Africa, and New Zealand. It has been known to attack man in Europe, Africa and Asia, and may produce a serious form of wound myiasis. The young larvae feed near the surface, but older larvae may bore deeply into healthy tissue. Onorato, an Italian, recorded cases of infestation of the ear and sinuses which were not previously diseased. Other cases of auricular myiasis have been recorded. The virulence of different strains varies. A Chinese strain is said to be

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particularly serious, whereas in America this species seems to confine its attacks to diseased tissue; in fact, it is the species most commonly used in wound therapy. Records of this species in enteric myiasis have been published, but they should not be accepted without further substantiation."

The larva is equipped with powerful mouth hooks which enable it to burrow deeply into the aural and nasal cavities, devouring in its passage all structures — including mucous membrane, muscle, cartilage, periosteum, and even bone. This accounts for the fatalities observed. Dr. H. Marshall Taylor[2] reports a death within four days from meningitis. In a series by Yount and Sudler[3] 23 deaths are reported. Aubertin and Buxton[4] report 179 cases with 15 deaths. Harris[5] reported two deaths out of five cases.

In human myiasis, by far the majority of deaths have occurred in the nasal infestations. The fly seems to be attracted by the fetid odor frequently found in cases of ozena, chronic rhinitis, syphilitic disease, epithelioma, bleeding perforations of the septum, etc.

Men that sleep outdoors in hot weather comprise most of the cases. The fly crawls in more or less unmolested and lays its eggs. These usually hatch within 24 hours and the larvae start feeding. Other flies may repeat the performance and a continuous cycle of infestation may be kept up before the patient realizes the seriousness of his condition.

We, in the Central and Northern states, might consider ourselves immune from the occurrence of myiasis, yet the possibility is there, when we are told that these flies may migrate as far as 1,500 miles north of their own wintering zone.

Patients with otitis media, when living around or near cattle and sheep farms, should be warned to wear cotton in the ear canal. Patients with vulnerable and predisposing nasal conditions should be under periodic observation and treatment.

CASE REPORT.

W. C., male, age 41, was seen in my office September 4, 1952. He gave history of sharp piercing pain in the right ear occurring the day before and persisting through the night. On arising the ear was bleeding but there was no relief from pain; in fact, when seen about 10 a.m. patient was

extremely nervous and walking the floor. On examination the ear was filled with bloody serum and, therefore, it seemed strange that he should have no relief from suffering. After cleaning out the canal, the entire drum membrane seemed to be in a state of active vibration; on careful examination a white wavy object seemed to protrude through the constant pouring of serum. With an ear forceps this was grasped and pulled out, proving to be a live, active maggot. On looking again, a second one appeared. Then it was seen that there was a perforation occupying one third of the drum membrane and other maggots were down in the middle ear, feeding. They were extremely active and in a split second would duck out and back through the perforation. After about twenty minutes work they were caught one by one and removed with the forceps until a total of five were taken out. Each one measured 13 mm. in length. At this time no further vibration was seen and exudation of serum subsided. Patient's pain was completely relieved.

The ear drained for about ten days but finally cleared and when seen one month later the drum was healed over completely and hearing had returned to normal.

Delving into this man's history, I found that he was quite an alcoholic; that he worked in a factory and had a habit of lying down on the table during his lunch hour each day. Evidently while asleep and probably numbed from liquor, a fly had crawled into his ear and laid eggs. These flies as a rule choose an open wound or an area where pus is present; however, in this case I believe these maggots hatched out and attacked a perfectly healthy tympanic membrane and middle ear. This conclusion is reached because the ear returned to normal and, secondly, the patient disclaimed any history of faulty hearing, running ear or middle ear abscess.

One of the maggots was saved alive, later examined carefully by Emil Kotcher, Sc. D. (Associate Professor of Microbiology), of the School of Medicine of the University of Louisville, and was identified by examination of the stigmal plate as the larva of the green-bottle fly of the species *Phaenicia sericata* (Meigen). This is one of the principal sheep-maggot flies, and the following is worthy of note: During 1950 and 1951 the Communicable Disease Center of the U. S. Public Health Service[6] and the Kentucky State Department of Health carried on a program of investigation and fly control measures in Harlan, Kentucky. Of 2090 flies trapped and examined 9.7 per cent were of the species *Phaenicia sericata,* and of the *Calliphoridae* this was the second most common.

CONCLUSION.

1. This report deals with the danger of occurrence, although a rarity, of myiasis in man as well as animal live stock.

2. The condition may result fatally if not recognized and immediately eradicated.

3. A case is cited of infestation of a normal ear with the green-bottle fly worm *(Phaenicia sericata)* with symptoms, treatment and subsequent return to normal.

4. Even though not used in this case, the larvae are readily killed by concentrated chloroform vapors or benzol.

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HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

June 1, 1953.

Audicon Models 400, 415, 530 and 615.

Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Auditone Model 11.

Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.

Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3; Dahlberg Model D-4.

Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 149 Church St., New York 7, N. Y.

Fortiphone Models 20-A and 21-C.

Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J; Maico Top Secret Model L.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Classic Model T9; Microtone Model T10; Microtone Model T612; Microtone Model 45.
Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Cub Model C; National Cub Model D (Duplex); National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.
Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Normatone Model C.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.
Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-2; Otarion Model G-3; Otarion Model H-1.
Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model Y (YM, YC and YC-7) (Veri-Small).
Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp.

Rochester Model R-1; Rochester Model R-2.

Manufacturer: Rochester Acoustical Laboratories, Inc., 117 Fourth St., S.W., Rochester, Minn.

Silvertone Model J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal; Model Cameo.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).
Manufacturer: A. M. Brooks Co., 64 S. Bonnie Brae St., Los Angeles 5, Calif.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Carl H. McCaskey, 608 Guaranty Bldg., Indianapolis, Ind.
Vice-Chairman: Dr. Fred W. Dixon, Rose Bldg., Cleveland, Ohio.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James M. Robb, Detroit, Mich.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 11-17, 1953.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., Oct. 5-9, 1953.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Herman J. Moersch.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Victor Alfaro.
Vice-Chairman: Dr. Irvin Feldman.
Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

OTOSCLEROSIS STUDY GROUP.

President: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Secretary: Dr. Lawrence R. Boies, Med. Arts Bldg., Minneapolis 2, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 11-17, 1953.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis, Ind.
President-Elect: Dr. Albert D. Ruedemann, 1633 David Whitney Bldg., Detroit 26, Mich.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., Oct. 16, 1953.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Justo M. Alonso, Montevideo.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fourth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. Ricardo Tapia Acuna, Mexico City.
Time and Place: Feb. 28 to Mar. 4, 1954, Mexico City.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Daniel L. Sexton, St. Louis, Mo.
President-Elect: Dr. John I. Marker, Davenport, Iowa.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.
Meeting:

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Peter N. Pastore, Richmond, Va.
President-Elect: Dr. G. S. Fitz-Hugh, Charlottesville, Va.
Vice-President: Dr. H. L. Mitchell, Lexington, Va.
Secretary-Treasurer: Dr. L. B. Sheppard, 301 Medical Arts Bldg., Richmond, Va.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Orwyn H. Ellis, M.D.
Secretary-Treasurer: Harold Owens, M.D.
Chairman of Section on Ophthalmology: Robert A. Norene, M.D.
Secretary of Section on Ophthalmology: Sol Rome, M.D.
Chairman of Section on Otolaryngology: Leland R. House, M.D.
Secretary of Section on Otolaryngology: Max E. Pohlman, M.D.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:00 P.M., fourth Monday of each month from September to June, inclusive—Otolaryngology Section; 6:00 P.M., first Thursday of each month from September to June, inclusive—Ophthalmology Section.

THIRD LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

President: Dr. Franz Conde Jahn.
Vice-Presidents: Drs. Julio Garcia Alvarez, Angel Bustillos and Celis Perez.
Secretary General: Dr. Victorino Marquez Reveron.
Secretary of Assemblies: Dr. Cesar Rodriquez.
Time and Place: Caracas, Venezuela, July 31, 1954.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Harry Newert, 555 Park Ave., New York (21), N. Y.
Secretary: Dr. Louis Joel Fleit, 66 Park Ave., New York (16), N. Y.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. William Banks Anderson, Durham, N. Car.
Secretary and Trasurer: Dr. Geo. B. Ferguson, Durham, N. Car.
Meeting: Charleston, S. Car., Sept. 13-16, 1953.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Clay W. Evatt, Charleston, S. Car.
Vice-President: Dr. David S. Asbill, Columbia, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. Car.
Meeting, Joint: Charleston, S. Car., Sept. 13-16, 1953.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.
Meeting: Honolulu, 1954.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Isaac H. Jones, 635 S. Westlake, Los Angeles, Calif.
Treasurer: Dr. Pierre Violé, 1930 Wilshire Blvd., Los Angeles, Calif.
Program Chairmen:
Otolaryngology: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.
Mid-Winter Clinical Convention annually the last two weeks in January at Los Angeles, Calif.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Harry P. Schenck.
Vice-President: Dr. William J. Hitschler.
Treasurer: Dr. Chevalier L. Jackson.
Secretary: Dr. John J. O'Keefe.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. M. Valentine Miller, Dr. Charles E. Towson, Dr. Thomas F. Furlong, Dr. Benjamin H. Shuster, ex-officio.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Vice-Chairman: Dr. K. W. Cosgrove, 111 E. Capitol Ave., Little Rock, Ark.
Secretary: Dr. F. A. Holden, Medical Arts Bldg., Baltimore, Md.
Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

CANADIAN OTOLARYNGOLOGICAL SOCIETY
SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. D. E. S. Wishart, 170 St. George St., Toronto, Ontario.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place:
Time:

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Victor M. Noubleau.
Secretary: Dr. Héctor R. Silva.
1o. Vocal: Dr. Salvador Mixco Pinto.
2o. Vocal: Dr. Daniel Alfredo Alfaro.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretario del Exterior: Dr. Juan Manuel Tato.
Sub-Secretario del Exterior: Dr. Oreste E. Bergaglio.
Secretario del Interior: Dr. Eduardo Casterán.
Sub-Secretario del Interior: Dr.Atilio Viale del Carril.
Secretario Tesorero: Dr. Vicente Carri.
Sub-Secretario Tesorero: Dr. José D. Suberviola.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presdente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Fernand Eeman, Ghent, Belgium.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting:
Time and Place:

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

President: Dr. Joao Penido Burnier.
First Secretary: Dr. Gabriel Porto.
Second Secretary: Dr. Roberto Barbosa.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. F. J. Monteiro Sales and Dr. Jose Martins Rocha.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Diaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asis R., Dr. Jorge Bergallo Yofre.

BUENOS AIRES CLUB OTORINOLARINGOLOGICO.

Presidente: Dr. Alberto P. Haedo.
Vice-Presidente: Dr. V. R. Carri.
Secretario: Dr. Renoto Segre.
Pro-Secretario: Dr. Carlos A. Gutierrez.
Tesorero: Dr. J. M. Tato.
Pro-Tesorero: Dr. Norberto Von Soubiron.

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tresoreo: Dr. Mario Arenas A.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

CONTENTS